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POST-IRRADIATION INFARCTION OF THE FEMORAL HEAD

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It is widely accepted that irradiation can damage bone, but the exact mechanism by which the process occurs is not fully understood. In those bones which are most frequently affected as a result of heavy irradiation, e.g. the jaw, infection is often superadded and pure radiation changes are difficult to unravel from those of superadded infection.

In bone situated deeply in the soft tissues, far removed from sources of potential infection, the 'pure' effects of radiation on bone may be more clearly observed. The femoral head and neck are constantly subjected to relatively heavy irradiation in justifiable therapeutic attempts to obtain adequate dosage in malignant conditions involving the pelvis. Here, the consequence of heavy therapeutic dosage is paid in the post-irradiation fractures of the neck of the femur. Various authors—Gratzek *et al.* (1945), Hight (1941), Kalayjian (1938), Kok (1953), Kulseng-Hanssen (1946), MacDougall *et al.* (1950), McCrorie (1950), Okrainetz and Biller (1939), Peck (1939), Ruther (1953), Slaughter (1942), Smith (1954), Smithers and Rhys-Lewis (1945), Stampfli and Kerr (1947), Truelsen (1942)—have recorded series of cases where the frequency of this complication has been recorded as being between 0·1 and 3·2% (Vaughan, 1955). But despite the frequency of these changes in the bones themselves no mention has been made in these reports of any changes in the articular surfaces of the joints in the irradiated areas.

The purpose of this communication is to record an example of a localized infarction of the femoral head occurring as a result of heavy therapeutic irradiation unavoidably delivered to this structure in the treatment of a metastatic deposit in an adjoining bone.

CASE REPORT

Mrs. A.B., a female aged 51 years, had been operated on 7 years previously for an enlargement of the thyroid gland with a retrosternal extension. Five years later, she consulted a physician for tiredness and was given ½ gr. of thyroid daily, with improvement. She had remained free from any symptoms until 1 year later, when she suffered slight pain in the right hip when ascending stairs. At this time physiotherapy was prescribed, with some relief of her symptoms. One year later her symptoms recurred and she was X-rayed at this time and an osteolytic deposit was seen in the right ischium.

On clinical examination, she was found to be well nourished and in considerable pain. There was limitation of movement in the right hip with spasm of the surrounding muscles. There were no glands palpable nor was the liver or spleen palpable.

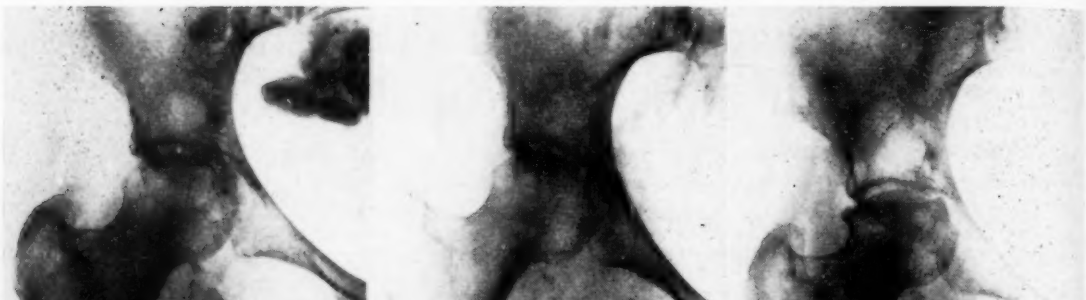
Radiographic examination showed damage to the right ischial bone, with extensive destruction of the ascending ramus of that bone as far as the acetabulum. The bony architecture of the head and neck of the femoral shaft was normal. A radio-active iodine study revealed an increased uptake over the lesion, which was considered to be a metastatic deposit from a malignant thyroid tumour.

Deep X-ray therapy (220 kv. with a Thoraeus filter) was administered from 11 January 1955 to 25 February 1955, a total tumour dose of approximately 5,000 r. being delivered. Partial, but not complete, relief of the symptoms was achieved. In view of continuing symptoms a second course of deep X-ray therapy was commenced on 4 April 1955 and continued to 25 April 1955 when a further total estimated tumour dose of 2,864 r. was administered. A therapeutic dose of 50 mc. radio-active iodine was also given at this time. A radiograph of the right hip joint on 5 April 1955 revealed some slight re-formation of bone within the deposit, but in the ascending ramus of the ischium little regeneration of the new bone had occurred.

The patient remained relatively free of pain but still suffered from some limitation of movement of the right hip. A radiograph of the right hip joint on 27 August 1955 showed a collapse of the articular surface of the femoral head with a loose separated fragment of bone similar to an osteochondrosis dissecans affecting more than one-third of the femoral head. No history of any trauma was obtained and a review of the films taken on 5 April 1955 revealed that the portion of the femoral head showed an increase in density, suggesting an aseptic necrosis—a feature not appreciated at the time of examination.

DISCUSSION

The effects of radiation on bone have been the subject of much investigation. Ewing (1926) classified the changes into 4 stages depending on the degree of radiation. The first stage is a proliferative osteitis, the second stage is a stage of osteo-



Antero-posterior views of the right hip joint demonstrating the development of the area of osteochondritis in the femoral head. The metastatic deposit in the right ischial bone is clearly seen.

sclerosis, the third stage with increasing dosage is an aseptic necrosis with the formation of sequestra, and the last stage is that of a superimposed infection. Ewing (1926) also considered that the secondary irradiation from the inorganic elements affected the organic elements of the bone.

More recently Kok (1953) and Stampfli and Kerr (1947) have stressed that the damage to the blood supply may be one of the causes responsible for the changes in the bone. The persistent dilatation of the vessels in the Haversian canals results in absorption with consequent weakening of the bone. Likewise Truelsen (1942) commented on the absence in histological sections of irradiated bone of osteoclasts and osteoblasts, considering that these features were the result of damage to the vessels although the internal coats of the vessels were intact.

Furthermore, as Stephenson and Cohen (1956) point out in a discussion on post-irradiation fractures of the neck of the femur, in the majority of their series healing of the fractures occurred readily, a feature which would hardly be expected if the blood supply was impaired. In one of the cases in their series, however, heavy dosage of irradiation resulted in necrosis of the femoral head with no apparent healing.

In the findings in post-irradiation fractures of bone it is probable that the changes noted are proportionate to the dosage of irradiation received by the bone. With the lesser degrees of dosage the cellular elements of the bone are probably affected, whilst with heavier dosage changes due to vascular disturbances also play a part in the changes that occur.

Little has been mentioned in the literature about the effect of radiation on joints as opposed to bone, but it is possible that in analogy to bone the cellular elements are destroyed with the lesser degrees of dosage whilst with the heavier dosages the vascular elements are involved.

In the case presented the heavy therapeutic dosage necessarily delivered to the bone resulted in an unusually high dosage being received by the femoral head with the subsequent development of an avascular necrosis and an infarction of the femoral head.

SUMMARY

1. A case showing the appearances of an osteochondritis dissecans of the femoral head is described.

2. It is postulated that these appearances are the result of infarction of the femoral head following heavy irradiation therapy.

3. The mechanism of the infarction is discussed.

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VAN DIE REDAKSIE

HORMOONBEHEER VAN BORSKANKER

Die begrip van 'n hormoon-afhanklike gewas is nie nuut nie. Meer as 60 jaar gelede is die eierstokke van 2 pasiënte met gevorderde kanker van die bors verwyder met heilsame gevolge,[1] en vir baie jaar daarna is terapeutiese kastrasie sporadies uitgevoer. Lacassagne se eksperimentele bewys dat neoplastiese veranderinge in die borsweefsels van muise, wat op groot hoeveelhede estrogeen gevoer is, teweeggebring kan word, is gedurende die afgelope jare gesteun deur die ontwikkeling van borskanker by manlike pasiënte wat met estrogene vir kanker van die prostaat behandel is.[2] Indien die kanker deur die estrogeen bevorder is, is dit 'n logiese afleiding dat die kanker sal afneem, of ten minste gestuit sal word, as hormoonproduksie ingekort word. Dit kan nie deur ooferektomie alleen gedoen word nie, nie eers deur ooferektomie gevolg deur adrenalektomie nie, want dit is gevind dat estrogeenuitskeiding—alhoewel dit onbestendig en spasmodies is—uiteindelik amper altyd weer hervat.[3] Dit lyk of hipofisektomie self nie eers estrogeen uit die urien verwyder nie.[4] 'n Rede hiervoor mag wel wees dat die chirurgiese neutralisasie van die besondere kliere onvolledig is, maar dit skyn verkeerd te wees om dit te aanvaar, en die moontlikheid dat ander bronne van estrogeen mag bestaan, moet ondersoek word. Miskien produseer die gewas self hormone, alhoewel dit bekend is dat terugwyking in die aanwesigheid van aanhoudende estrogeenproduksie kan geskied.

Wat ookal die teoretiese en eksperimentele beskouings mag wees, is die hormoonbeheer van borskanker 'n agtenswaardige en algemeen-aangenome behandelingsmetode. By Guy's Hospitaal, Londen, het ooferektomie met algehele adrenalektomie teen die einde van 1953 die standaardbehandeling geword vir pasiënte met onopereerbare kanker wat nie geskik was vir radioterapie en chirurgie nie, en wat nie op hormone gereageer het nie. Tot op daardie tydstip is 18 gevalle op hierdie wyse behandel met wisselende sukses. Dit is gevind dat gekombineerde adrenalektomie beter resultate gelewer het as net ooferektomie. Intussen het hipofisektomie op die voorgrond getree as die alternatiewe operasie. Luft en sy Sweedse span het 41 gevalle van borskanker, op wie hierdie operasie uitgevoer is, aangemeld. Twee-en-twintig van hulle (54%) het objektiewe afname, wat ongeveer 18 maande geduur het, getoon—slegs een vir langer as 2 jaar.[5] (Luft bepaal ‚objektiewe afname' as krimping van sigbare, voelbare of radiologiese metastases, of toenemende beendigtheid sonder toename van die ou metastases, of voorkoms van nuwes.) Terwyl ander navorsers naasteby 'n ooreenstemmende graad van terugwyking aangemeld het (Pearson *et al.* 51% van 41 gevalle;[6] Kennedy 64% van 28 gevalle[7]), het hulle nie dieselfde mate van sukses gehad as die Swede met die duur van die afnameperiode nie.

EDITORIAL

HORMONAL CONTROL OF MAMMARY CANCER

The concept of a hormone-dependent neoplasm is not new. More than 60 years ago the ovaries of 2 patients with advanced cancer of the breast were removed with beneficial results,[1] and for many years thereafter therapeutic castration was practised in a sporadic manner. Lacassagne's experimental evidence of producing neoplastic changes in the mammary tissue of mice fed on large amounts of oestrogen has been supported in recent years by the development of breast carcinoma in male patients on oestrogens for carcinoma of the prostate.[2] If the cancer is promoted by the oestrogen, a logical deduction is that the cancer will regress, or at least be halted, if hormone production is curtailed. This cannot be done by oophorectomy alone, nor even by oophorectomy followed by adrenalectomy, for it has been found that oestrogen excretion—though variable and spasmodic—is nearly always eventually re-established.[3] Even hypophysectomy itself does not appear to eliminate oestrogen from the urine.[4] A reason for this may well be that the surgical neutralization of the particular glands is incomplete, but it seems wrong to assume this, and the likelihood that other sources of oestrogen may exist must be examined. Perhaps the tumour itself produces hormones, although it is known that regression can occur in the presence of continuing oestrogen production.

Whatever the theoretical and experimental considerations, the hormonal control of mammary carcinoma is a reputable and widely-accepted line of treatment. At Guy's Hospital, London, oophorectomy with total adrenalectomy had become by the end of 1953 the standard practice in patients with inoperable cancer who were unsuitable for radiotherapy and surgery, and who had not responded to hormones. By that date 18 cases had been treated in this manner with variable success. It was found that combined adrenalectomy gave better results than oophorectomy alone. Meanwhile hypophysectomy had come to the fore as the alternative operation. Luft and his Swedish team reported 41 cases of breast cancer upon which it had been carried out, 22 of which (54%) showed objective remissions which lasted about 18 months—only one longer than 2 years.[5] (By 'objective remission' Luft denotes shrinkage of visible, palpable or radiological metastases, or increased bone density without progression of the old metastases, or appearance of new ones.) Whilst other workers reported a roughly

By nie een van die reekse het die gemiddelde terugwyking langer as 7½ maande geduur nie. Nietemin reken die span van Guy's Hospitaal dat hulle gehipofisektomeerde pasiënte oor die algemeen 4 maande langer as die geadrenalektomiseerde pasiënte gelewe het. Inderdaad is Atkins en sy kollegas, waar hulle die resultate van die twee metodes sorgvuldig vergelyk, skynbaar ten gunste van hipofisektomie, alhoewel hulle beklemtoon dat die operasie statisties nie die oorhand oor adrenalektomie het nie.

Watter geval behoort aan operasie onderwerp te word? En aan ooferektomie met adrenalektomie, of aan hipofisektomie? Soos die *Lancet* skryf, is ‚daar nog geen bevredigende wyse om te voorspel watter pasiënte met borskanker op adrenalektomie of op hipofisektomie sal reageer nie'.[8] Luft *et al.* doen aan die hand dat ouer pasiënte, veral vrouens by wie die maandstonde opgehou het, waarskynlik minder bevredigend sal reageer. Eweneens word dit verklaar dat metastases in die lewer of brein met 'n betreklik langdurende bestaan, prognosties van slegte betekenis is. Die mening is dat terapeutiese kastrasie 'n goeie aanduiding van die moontlike uitwerking van hipofisektomie gee; as die pasiënt op ooferektomie reageer, is dit moontlik dat sy ook op hipofisektomie sal reageer. Maar hierdie is slegs kliniese aanwysers en spesifieke aanduidings vir operasie was baie moeilik om vas te stel. Tot onlangs kon die patoloog baie min nuttige inligting verskaf. Daar is geen wederkerige betrekking tussen tellings van mitotiese selle en reaksie op behandeling nie. Uitskeiding van estrogeen en gonadotrofien in die urien word nie beïnvloed nie. Kalsiumvlakke in die urien is nuttig slegs as osteologiese metastases aanwesig is—'n daling van die vlak na 'n inspuiting van estrogeen en 'n styging na kortisoon, doen aan die hand dat die gewas hormoon-afhanklik is en dat adrenalektomie van nut sal wees. Vroeër vanjaar het die kliniese patoloog by Guy's Hospitaal die aandag gevestig op 'n betekenisvolle verskynsel wat wel mag bewys dat dit 'n oplossing mag bied vir watter gevalle vir operasie gekies moet word. Merivale *et al.* het die breuke van algehele neutrale 17-ketosteroïede in die urien van 15 gevalle van borskanker, voor en na operasie (òf hipofisektomie òf adrenalektomie met ooferektomie) bestudeer. By hierdie gevalle is die verhouding van 11-deoksi-17-ketosteroïede tot 11-suurstofhoudende (of -gehidroksileerde) -17-ketosteroïede geskat. By die 11 gevalle wat klinies deur die operasie gebaat is, is dit gevind dat hierdie verhouding groter as 1 is, en dat die uitskeidingspatroon met dié wat by gesonde persone gevind word, ooreengekom het. By die 4 gevalle waar daar geen afname was nie, was die verhouding minder as een. Terwyl die vertolking wat hierdie navorsers aan hulle bevindinge heg, baie behoedsaam is, is dit moontlik dat hierdie proefneming mag toon dat dit op 'n praktiese wyse van nut is, des te meer daar hormoonbeheer vir die eindstadium van borskanker 'n gevestigde entiteit is.

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corresponding regression rate (Pearson *et al.* 51% of 41 cases;[6] Kennedy 64% of 28 cases[7]) they do not share the Swedes' success over the duration of the period of regression. In neither series was the average regression of longer duration than 7½ months. Even so, the Guy's Hospital team consider that their hypophysectomized patients survived on the average 4 months longer than the adrenalectomized ones. In fact, Atkins and his colleagues in their careful comparison of the results of the two procedures seem to favour hypophysectomy, although they emphasize that the operation has no statistical superiority over adrenalectomy.

Which case should be submitted to operation? And to oophorectomy with adrenalectomy, or to hypophysectomy? As the *Lancet* writes, 'There is still no satisfactory means of predicting which patients with breast cancer will respond to adrenalectomy or to hypophysectomy.'[8] Luft *et al.* suggest that older patients, particularly post-menopausal women, are less likely to respond satisfactorily. Similarly, metastases in the liver or brain with a relatively long-standing growth are said to be of bad prognostic significance. Therapeutic castration is believed to give a good indication of the likely effect of hypophysectomy; if the patient responds to oophorectomy, she is likely to do so to hypophysectomy. But these are only clinical pointers, and specific indications for operation have been hard to determine. Until recently the pathologist could give little useful information. There is no correlation between counts of mitotic cells and response to treatment. Excretion of oestrogen and gonadotrophin in the urine is unaffected. Calcium urinary levels are useful only if osteolytic metastases are present—a fall in the level with an injection of oestrogen and a rise with cortisone suggests that the tumour is hormone-dependent, and that adrenalectomy would be of benefit. Earlier this year the clinical pathologist at Guy's Hospital drew attention to a significant clinical phenomenon that may well prove the key to the selection of cases for operation. Merivale *et al.* have studied the fractions of total neutral 17-ketosteroids in the urine of 15 cases with breast cancer, before and after operation (either hypophysectomy or adrenalectomy with oophorectomy). In these cases the ratio of 11-deoxy-17-ketosteroids to 11-oxygenated (or -hydroxylated) -17-ketosteroids was estimated. In the 11 cases which had benefited clinically by the operation, this ratio was found to be greater than 1, and the excretion pattern resembled that found in healthy subjects. In the 4 cases where no remission occurred, the ratio was less than one. Whilst the interpretation that these workers place upon their findings is very cautious, it is possible that this test may prove to be useful in a practical way. This is the more so since hormonal control of terminal mammary cancer is an established entity.

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A NEW DERMATOME

The problem of burns looms relatively larger as the hospital stay of other surgical conditions becomes reduced. The average time spent in hospital by cases of herniotomy, appendicectomy and cholecystectomy is usually less than one week, and even an ulcer-gastrectomy need rarely keep a patient in bed for more than the same time, but the burn—more often than not in a child—occupies a bed for weeks and months. Anything, therefore, that can be done to reduce this time will be amply rewarded not only by prevention of suffering, but also in reduction of hospital charges. Late sequelae such as contractures will also be reduced because the sooner healing is achieved, the less fibrosis will develop; once the wound is covered with skin, fibrosis is halted and rehabilitation can commence.

The surface of the average burn or scald is composed of areas of varying severity whose exact position it is not possible to predict in the first few days. Scattered over most burnt areas are islets of surviving epithelium and this damaged epithelium offers very little resistance to further injury and is easily completely destroyed by infection which develops on the burnt areas and effectively kills these islets. In the recent past the main aim of therapy, once the initial state of shock and the later stage of fluid and protein loss are safely passed, has been to prevent infection and so to preserve these islets.

Since epithelium grows centrifugally, even a light peppering of skin soon coalesces, and very large areas can quickly be covered. This is the explanation of the rapid healing of large areas of burnt skin that are occasionally reported in advertisements of proprietary ointments; the depth of the burn has been superficial, the many islets have coalesced and healing has taken place.

But with all the care to prevent infection a large number of cases are referred to hospital and occupy beds. It has long been recognized that the best dressing for these areas is autogenous skin applied as soon as the infection is under control. Many practitioners, in relatively isolated areas, are well aware of this, but have not been able to acquire the skill and experience necessary to cut these thin split-skin grafts. To overcome this difficulty, mechanical dermatomes of greater or lesser complexity have been invented and described from time to time. Davies and Davies, on this page of the *Journal*, describe a new dermatome which has several advantages. It is inexpensive, simply constructed, portable and safe and requires practically no skill or experience to ensure a good cut. With very little skill in cutting Thiersch grafts it will allow a practitioner, operating in any small hospital, when he feels that a burnt area is ready to be grafted, to cover the granulations with strips of skin and to feel relatively sure that within 14 days healing will be complete. The instrument works on the principle of the barber's hair-clipper. A standard safety-razor blade, which is easily replaced, moves a few millimeters from side to side to achieve the cut. The use of this instrument should have a noticeable effect in shortening the stay in hospital and reducing the morbidity in these difficult cases.

MIDGET DERMATOME AND STRIP SKIN GRAFTING

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The proposal for yet another dermatome calls for a few words of justification.

The need for this dermatome originated in the wish to speed up the skin coverage of extensive burns, particularly in children. Nowadays, the surgeon is faced with burns of such extent as formerly would never have lived to need grafting, but, the patient having been brought through to this stage, skin covering should now be carried out quickly and completely. The aim must be complete coverage by the end of the 4th week. Delay in skin grafting is due chiefly to the difficulty of getting sufficient skin in these very sick patients, and the result is that grafting is only completed after the seeds of future contractures have been sown and the soil has become unkindly to grafts. Particularly is this so where the surgeon does not possess special guarded skin-graft knives or is inexperienced in freehand skin cutting. Even with guarded knives the difficulties of taking skin in large quantities tend to make the surgeon shirk the operation.

At one time we sought a solution in the use of homografts such as parental skin, but though helpful, this was far from ideal, and we have always been working towards our ideal of complete coverage with overlapping strips of the patient's own skin.

Our object in cutting the skin into strips is not to economise skin but to ensure take. On infected granulating areas, strips survive more surely than larger areas of skin would. On freshly excised areas, no matter how careful the haemostasis, haematoma formation is frequent, and if such an area is covered by one large single graft, the graft may be lifted up into a tent by clot, with resulting failure to take. The danger is minimized where the skin is put on in strips. Strips allow blood and secretions to escape at the seams, leaving the grafts in place. It is very important to get granulating areas completely covered at the first grafting; a second grafting to cover areas left between grafts does not give nearly as good a result as the first. Many of the new patches fail to take, and the skin from near-by strips seems unable to grow uphill on to the rising granulation tissue.

This means that a lot of skin is needed if complete coverage is to be carried out at the first operation, and not too much

time should be spent at operation in taking it; some quick mechanical form of cutter is necessary.

We have watched great artists operating with many forms of hand cutting knives, guarded and unguarded, but have always thought, even while admiring them, that there surely must be some better and quicker way. With this in mind we experimented with all forms of dermatomes and concluded that the electrodermatome came nearest to the ideal.

There were, however, still certain objections. Firstly, the initial cost of the machine was high and, although the blades were called 'throw-away blades', when you consider that each one may cost 10s. 0d. to 12s. 6d. it becomes expensive, because we have never succeeded in satisfactorily using such a blade twice—or even once if it has been sterilized and put away without being used. Further, there is no means of restoring the edge by stropping. A second disadvantage of the usual dermatome blade, is its length which makes many areas inaccessible to operation with such a blade. In bad burns one rarely finds an area which will allow a graft to be taken the full available width of the blade. This is especially so in children, the convexity of whose limbs allows of grafts often only an inch wide.

In the face of these difficulties why attempt to take wide grafts? They should be cut into strips or stamps anyway, so that it would be time-saving to take them about an inch wide straight away. A dermatome with a cutting head this width at once opens up many new fields from which to take grafts; the pectoral regions, the abdomen, the strips over the erector spinae, the upper and lower arms and legs all become available, and the ideal of taking sufficient skin to cover the defect entirely, becomes practicable.

We have in fact found that with the narrow dermatome we can take skin from practically anywhere on the body and with very little practice it is possible to wander from abdomen or back, up over the ribs or across the abdomen at will. It is obvious what this means in a case of burns of say 40% of the body surface, where the legs, from which the grafts are usually taken, are burned and no longer available as donor areas.

Most of the urgent extensive grafts are for covering big burned areas with infected granulating surfaces, and for these areas skin strips are ideal. For almost all skin grafting we need enough skin for complete coverage, but in narrow strips of appropriate thickness, taken in such a way that the donor area heals so quickly that they may give new grafts if necessary within 3 weeks.

For these requirements the midget dermatome is ideal. It can do no damage, it can be adjusted for varying thickness, it is simple, speedy and economical. The blade we use is a

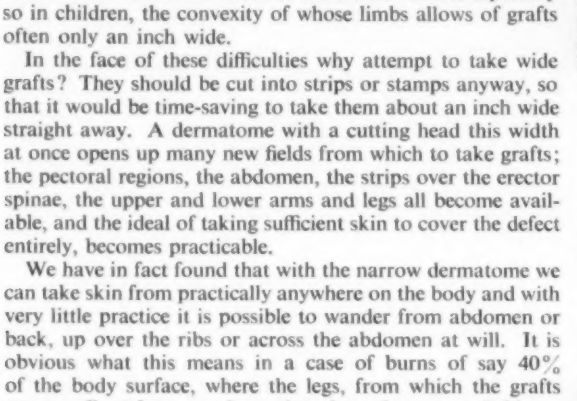

Fig. 1. Case A.B. Left leg showing area strip-grafted 7 days before, first dressing with grafts healed; donor area on right leg also healed.

Fig. 2. Case X.Y. Donor areas 2 weeks after grafting ready for second crop.

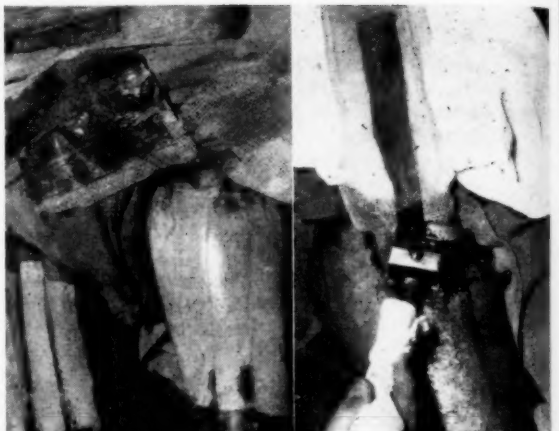

Fig. 3. Case X.Y. Second crop taken: strips shown on tulle gras.

Fig. 4. Case X.Y. Dermatome at end of cutting a strip.

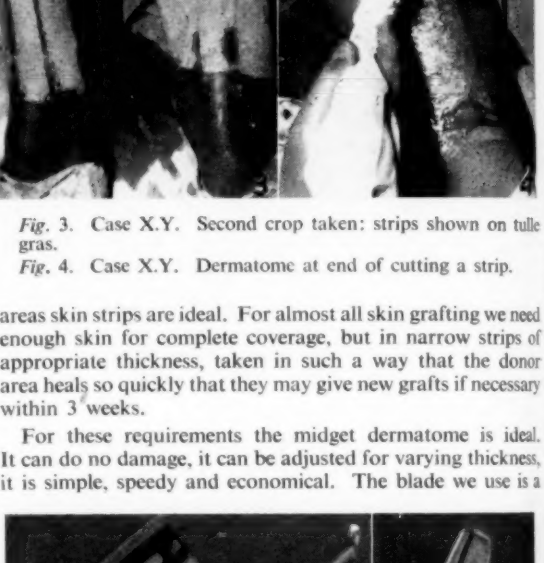

Fig. 5. Dermatome—open, showing razor blade in position.

Fig. 6. Dermatome—shut, ready for use.

commercial safety-razor blade of the injector type, costing a few pence each. One blade is sufficient for any but the very largest grafts. The simplicity of the machine is such that skin grafting can be done by every surgeon or general practitioner. With such a machine it is possible to cover completely very extensive burns in one operation, and to have the whole area healed by the end of the fourth or fifth week.

Burned cases, instead of being referred late to plastic units at the larger hospital could be dealt with by the general surgeons or general practitioners at their local hospitals, thus avoiding long hospitalization and the tragedy of old burn scars and contractures which will need further plastic treatment later.

The dermatome* illustrated (Figs. 4, 5 and 6) is built to fit like a dental hand piece on to the cable of a dental machine. The drive moves a small blade in a side-to-side cutting action. We have constructed the machine to take the 'Pal' injector-type safety-razor blade. The blade can be changed in seconds by turning back the safety lever, pulling back the collar, and pressing up the base plate. These movements are reversed to hold the blade in place. The hood ends in a guard bar in front, which keeps the blade cutting at the desired thickness. The thickness of cut may be regulated by turning the milled nut to the appropriate setting. One blade costing a few pence will cut several feet of a skin strip and will suffice for all but the biggest grafts. The machine may be sterilized by boiling, autoclaving or immersion in T.C.F. solution,* but we have found boiling in A.C.10 solution† best, in that it ensures lubrication as well as sterilization.

We should like to offer our deepest thanks to Mr. Holmgren for helping us to prove that the idea was feasible, and to Mr. Cecil S. Marks, Mr. Kenneth Wheeler and Mr. H. R. Cooke for many brilliant ideas and much precise work. Thanks are also due to Mr. B. Todt, of Groote Schuur Hospital, for the illustrations.

* Available from the Marks Engineering Works, 118 Buitengracht Street, Cape Town (patent applied for).

* A 2% solution of each of thymol, cresol and formalin in methylated spirits.

† A proprietary preparation of mixed oil supplied by the Raven Oil Co., 170 Piccadilly, London, W 1, and supplied by most dental suppliers.

HYDROCALYCOSIS AND CALYCEAL DIVERTICULUM

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Hydrocalycosis and calyceal diverticulum are related but not identical conditions. They both represent dilatations of the renal collecting system, but differ in their pathological and radiological features. Their etiology has not been conclusively established, nor their specific relationship to the formation of renal calculi. Their importance lies in diagnosis, particularly radiologically, for on this will depend the management of the cases and the avoidance of unnecessary surgical procedures.

Historical

Hydrocalycosis and calyceal diverticulum have long been recognized. Prather (1941) in an historical survey stated that a similar condition was described in 1841, albeit under a different name. Since that time the terminology has differed from author to author but the lesions described show essentially similar features. Watkins (1939) and Winsbury-White (1939) were the first to use the term hydrocalycosis, and Prather's description is the first reference to calyceal diverticulum.

Pathology

According to Newman, Lowman and Waters (1952), Moore (1950), Mathieson (1953), Ferguson and Ward-McQuaid (1955), and Prather, the essential features of hydrocalycosis and calyceal diverticulum are that they are dilatations of the renal collecting system lined by transitional epithelium, although Holm (1948) states that the lining membrane may be altered by infection or calculus formation.

Dorsey (1949) and Prather differentiate macroscopically between calyceal diverticulum and hydrocalycosis. The former consists of a dilatation distal to a minor calyx communicating with the minor calyx by a fine channel (Fig. 1B), the latter is a dilatation of the calyx itself (Fig. 1C). Moore further subdivides hydrocalycosis on the basis of the anatomical site (Fig. 1C and D). This author does not recognize calyceal diverticulum as described by Dorsey (Fig. 1B), and considers hydrocalycosis and calyceal diverticulum (i.e. dilatation of the minor calyx, Fig. 1D), to be synonymous. In addition Dorsey differentiates pathologically between these two conditions and other localized cystic dilatations, such as congenital solitary cysts and cysts following obliterative pyelonephritis.

Etiology

The etiology of both these conditions has not been determined. Two main theories of their causation are postulated:

(*a*) *Achalasia.* Moore, Prather, and Watkins consider them to be due to achalasia or spasm of the musculature in the region of the calyceal neck. The anatomy of these muscles has been described by Henle (1866) and the hydromechanics of the renal calyces has been discussed by Narath (1940). On the basis of these descriptions it would appear that the situation of the lesions will depend on the anatomical site of the muscles affected. For example, achalasia of the muscles of the calyceal neck will produce hydrocalycosis, while affection of the muscles of the renal pyramids will produce a calyceal diverticulum.

(*b*) *Mechanical Obstruction.* Ferguson, Ward-McQuaid and Dorsey postulate that this is due to fibrosis, which may be secondary to infection or calculus formation, and Hyams and Kenyon (1941) consider that a localized obliterative pyelonephritis may produce it. In dilatation associated with calculus formation, MacAlpine (1949) states that an

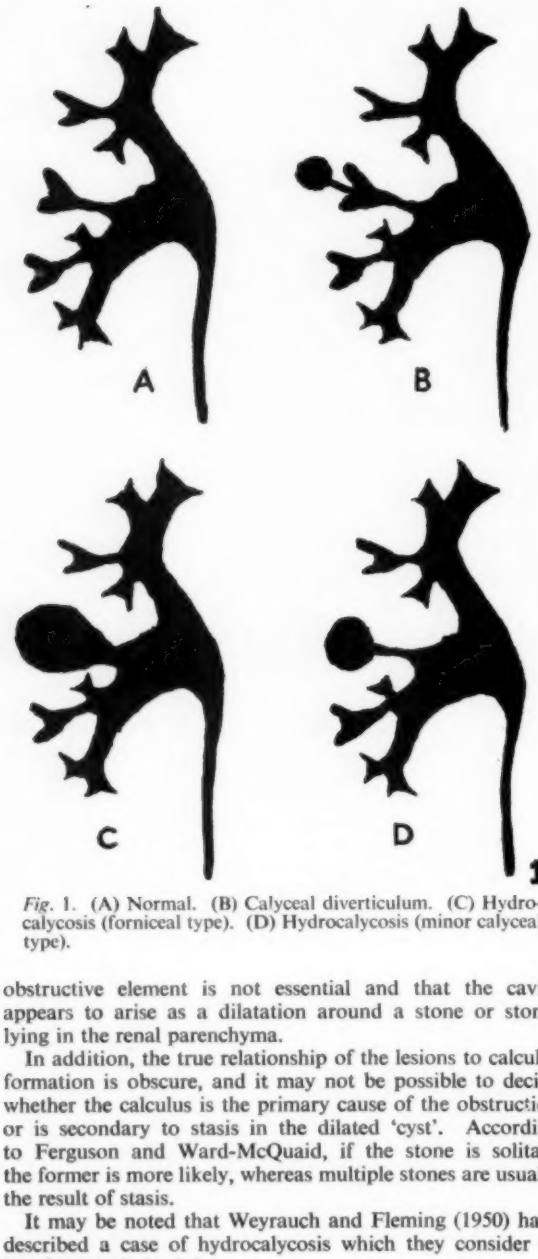

Fig. 1. (A) Normal. (B) Calyceal diverticulum. (C) Hydrocalycosis (forniceal type). (D) Hydrocalycosis (minor calyceal type).

obstructive element is not essential and that the cavity appears to arise as a dilatation around a stone or stones lying in the renal parenchyma.

In addition, the true relationship of the lesions to calculus formation is obscure, and it may not be possible to decide whether the calculus is the primary cause of the obstruction or is secondary to stasis in the dilated 'cyst'. According to Ferguson and Ward-McQuaid, if the stone is solitary the former is more likely, whereas multiple stones are usually the result of stasis.

It may be noted that Weyrauch and Fleming (1950) have described a case of hydrocalycosis which they consider to be of congenital origin.

Clinical Findings

The clinical findings are variable and no characteristic syndrome can be described. The most frequent symptoms are the following:

(*a*) Aching pain in the kidney region. Of the cases recorded in the literature this would appear to be the commonest symptom.

(*b*) Haematuria, with or without clot colic.
(*c*) Symptoms associated with infection and pyuria.
(*d*) Symptoms of calculus colic.

The conditions may be entirely asymptomatic.

CASE REPORTS

1. *Minor Calyx Hydrocalycosis.* A male aged 55 years, with a history of several attacks of left renal colic for 6 years, during the last 4 of which repeated X-ray examinations showed a dense calculus at the lower end of the left ureter without evidence of back pressure or dilatation of the calyceal system. Six weeks before the last pyelographic examination he passed a calculus

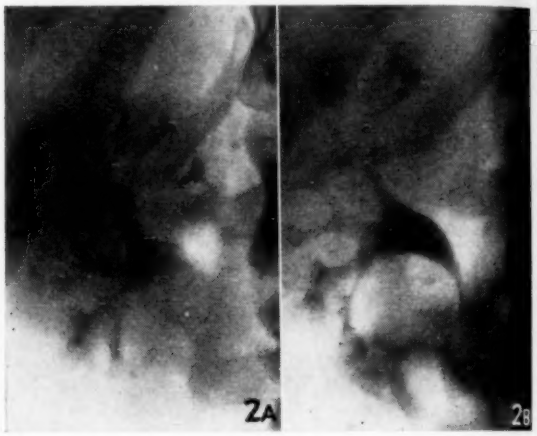

Fig. 2 (*a*). Case 1. IVP demonstrating minor calyceal hydrocalycosis in the upper pole of the right kidney. Retouched.
Fig. 2 (*b*). Case 1. IVP 4 years later. No appreciable change in the appearances.

painlessly and the films revealed that the stone was no longer visible. All films taken throughout the 4-year period showed a smooth, localized cavity containing contrast medium in the upper pole of the right kidney. This showed no change in size over 4 years. One of the upper minor calyces was not seen and the cavity containing contrast medium represented a dilated calyx (Figs. 2*a* and 2*b*).

2. *Calyceal Diverticulum.* A male aged 43 presented with a history of colicky pain in the left loin 5 days previously. This had lasted one day and was relieved by an injection. The urine was cloudy during the attack but there was no frank haematuria or dysuria. On the day before he was seen he felt a slight pain in the right loin similar to a pain experienced there 6 years previously. On that occasion the attack ended with the passage of a urinary calculus. Intravenous pyelography (Fig. 3) revealed a small cavity containing contrast medium distal to the calyces in the upper pole of the right kidney. A small channel could be seen communicating with one of the minor calyces. The calyces themselves showed no abnormality.

3. *Calyceal Diverticulum.* A female aged 44, first seen giving a history of an attack of severe pain in the left loin 2 months before. The attack lasted a few days and was relieved by aspirin. It coincided with an attenuated menstrual period. There was no dysuria and the urine was clear. Intravenous pyelography at that time was said to have shown a non-functioning left kidney and retrograde ureteric catheterization was performed. A repeat of the intravenous pyelogram 5 days later was said to have shown a normally functioning left kidney. Since that time the patient had been asymptomatic. Intravenous pyelography revealed a small cavity containing contrast medium distal to the calyceal system in the upper pole of the left kidney and communicating with a minor calyx by a fine channel. There was no other abnormality.

4. *Hydrocalycosis (forniceal type).* A middle-aged male suffered

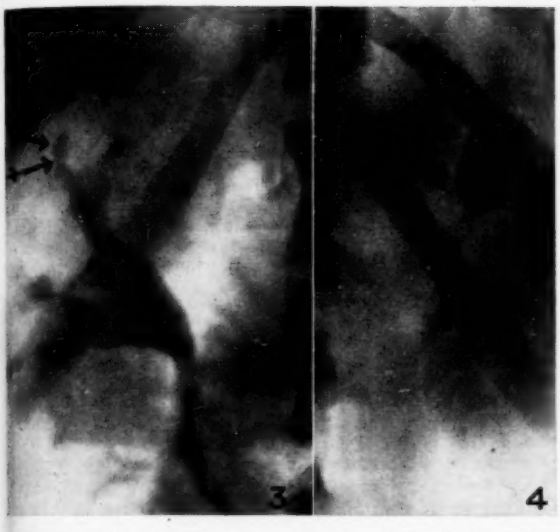

Fig. 3. Case 2. IVP. A small calyceal diverticulum(→) is shown, distal to a relatively normal minor calyx, with which it is connected by a fine channel (+ →).
Fig. 4. Case 4. Tomographic section of the left kidney during IVP, demonstrating a large hydrocalycosis of the forniceal type.

from recurrent urinary infections, which usually responded rapidly to antibiotics. There was no haematuria and no history of urinary calculus. Intravenous pyelography (Fig. 4) showed a large cavity containing contrast medium in the upper pole of the left kidney. This represented a minor calyx greatly dilated, distal to its junction with the major calyx. The patient was followed up over a number of years without alteration in the radiographic appearances and without clinical deterioration. Of late the attacks of infection have become less frequent.

DISCUSSION

The essential radiological feature in hydrocalycosis and calyceal diverticulum is a smooth, regular, round or oval cavity in the renal parenchyma which fills with contrast medium on intravenous or retrograde pyelography. The contrast medium tends to remain in the cavity for some time after it has drained from the rest of the collecting system. In calyceal diverticulum the cavity is distal to the minor calyx and communicates with it by a fine channel, whereas in hydrocalycosis the cavity represents a dilated calyx. This conforms with Dorsey's pathological description rather than with Moore's and is borne out by the appearances in the cases described. Features shown in cases 2 and 3 are not described by Moore (Fig. 3). The lesion is localized and the remainder of the collecting system of the kidney shows no abnormality; the kidney functions normally on intravenous contrast injection and the unaffected calyces show no distortion. In calyceal diverticulum the cavity tends to be rather small (cases 2 and 3) but in hydrocalycosis it may reach a considerable size (case 4). The lesions are not rapidly progressive and may be followed up for long periods, years in some cases, without apparent change.

None of the cases presented showed calculi directly related to the cavities in the renal parenchyma. However, cases 1 and 2 were associated with a history of calculus, although in case 1 the calculus was on the side opposite to the lesion and in case 2 the passage of the calculus predated the discovery of the lesion by 6 years. In case 3 no calculus was demonstrated but the history of calculus is strongly suggested. It would appear likely, therefore, that some definite relationship exists between hydrocalycosis, calyceal diverticulum and renal calculi.

As long as the existence of these conditions is recognized and their possiblity borne in mind, their radiological features are usually sufficiently characteristic to permit of an accurate diagnosis. However, as has been noted, their clinical features are variable and uncharacteristic, a finding borne out by the fact that only one case in the present series showed the symptom most commonly recorded in the literature (i.e. aching pain in the loin). For this reason the main responsibility in their diagnosis will rest with the radiological findings.

The differential diagnosis, therefore, particularly from a radiological standpoint, would appear to be important:

(*a*) *Tuberculous Lesions*. Tuberculous cortical abscesses in the earliest stages may present a picture similar to that of hydrocalycosis. However, untreated tuberculous lesions increase progressively in size and the degree of cortical involvement is considerably greater within a short period of time than in hydrocalycosis. In the later stages calcification may develop within the cortex. In addition, bacteriological studies of the urine may reveal the nature of the condition.

(*b*) *Cystic Lesions* include solitary congenital cysts and parapelvic cysts. Generally neither of these communicate with the renal collecting system and will not fill with contrast medium either on retrograde or intravenous pyelography. However, should a cyst rupture into a calyx or into the renal pelvis it may be indistinguishable radiologically from hydrocalycosis and the final diagnosis would depend on histological examination of the lining membrane. The cysts are lined by cuboidal epithelium in contradistinction to the transitional epithelium of the uninfected hydrocalycosis. Further, the cysts tend to produce considerably more distortion of the calyceal system.

(*c*) *Cystic areas* secondary to obstruction produced by an *obliterating pyelonephritis* will usually not fill with contrast medium, because they are cut off from the renal collecting system by the fibrous reaction. Should they fill they may be differentiated by a well-marked irregularity of outline, and a deformity of the associated minor calyces.

(*d*) *Tumours of the kidney parenchyma* produce calyceal distortion and, if large enough, deformity of the kidney outline itself. Even if they undergo cystic degeneration, it is uncommon for the cystic areas to fill with contrast medium on pyelographic examination.

(*e*) *Pyelo-Interstitial Reflux*. The contrast medium in the renal parenchyma has a rather hazy ill-defined margin in contradistinction to the clear-cut, well defined margin seen in hydrocalycosis or calyceal diverticulum. Also the contrast medium clears from the kidney substance relatively quickly.

(*f*) *Renal Papillary Necrosis*. The radiological picture may be similar to calyceal diverticulum but the patients are critically ill and the condition is commonly associated with diabetes mellitus or an obstructive uropathy, and always with pyelonephritis (Christoffersen and Anderson—1956).

Once the diagnosis has been made, in the absence of incapacitating symptoms, expectant treatment is the choice.

Recurrent infection may require intensive medical treatment for adequate control (case 4). However, if infection is persistent surgery becomes necessary, the majority of workers preferring conservative local resection rather than radical surgery.

Ferguson and Ward-McQuaid consider follow-up radiological examinations unnecessary where no complicating symptoms are present. Although cases 1 and 4 support this contention it is probably desirable to have one or perhaps two re-examinations at 3-monthly intervals to exclude the possiblity of a tuberculous lesion with certainty.

SUMMARY

1. A brief historical review of dilatations of the renal collecting system is presented.

2. The pathological changes are discussed with special reference to the differentiation of hydrocalycosis and calyceal diverticulum. Theories of causation are presented.

3. The variability of the clinical features is stressed and the importance of radiological diagnosis is emphasized.

4. Four cases are presented, 2 of hydrocalycosis and 2 of calyceal diverticulum, with radiological illustrations.

5. The differential diagnosis of these conditions and their relationship to renal calculus is discussed.

6. Management is briefly outlined.

I wish to thank Dr. Eric Samuel for his helpful advice and encouragement in the preparation of this paper. I am indebted to Mr. R. Campbell-Begg and Mr. I. Maisels for permission to publish the reports of cases 1 and 2.

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CONSTITUTIONAL HEPATIC DYSFUNCTION

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Constitutional hepatic dysfunction is a rare condition in which there is hyperbilirubinaemia without evidence of liver disease. This bilirubin gives an indirect van den Bergh reaction and the disorder is completely benign. Long-continued mild jaundice in a young person is usually considered to be due to chronic viral hepatitis if haemolytic disease can be excluded, but it is important to bear constitutional hepatic dysfunction in mind in the differential diagnosis. This is particularly so because bed rest features prominently in the management of viral hepatitis. These problems are illustrated by the case now presented.

CASE REPORT

In October 1956 a 29-year-old European University lecturer noted considerable fatigue and was exhausted at the end of each of his lectures, when there was some discomfort in the right side of the chest. This troubled him for another 6 weeks, when he developed a pyrexial illness that lasted a few days. His practitioner, noting that his urine was dark, found that he had mild jaundice, and made a diagnosis of infective hepatitis. It was after this that the patient became slightly nauseated—though he retained his appetite—and developed intermittent discomfort in the right upper quadrant. The stools were normal in colour and the urine was dark only for a few days, while the temperature was elevated. To the patient the illness was no different from attacks of influenza from which he had suffered in the past. The serum bilirubin was noted to be 2·2 mg.% (direct 1·3) at this time and 1·4 (direct 0·9) 2 weeks later.

In spite of spending 6 weeks in bed his symptoms persisted, and in February 1957 he again saw his practitioner, when the bilirubin was 2·0 (direct 0·7). Another 6 weeks in bed did not produce much improvement, but the symptoms were at no time severe.

He presented himself at Groote Schuur Hospital on 16 April 1957. Physical examination was quite negative, save for extensive lower-motor-neurone paralysis of the left arm, the result of an attack of poliomyelitis in 1947. Icterus was not detected clinically, and the urine was free of bile and urobilin, but the serum bilirubin was 4·0 mg.% with a negative van den Bergh reaction. Accordingly he was admitted for further study. Again physical examination was negative; in particular, the liver and spleen were not enlarged and there was no hepatic tenderness. These signs were unchanged after strenous exercise lasting 2 hours. The results of urine analysis were negative. Examination of the blood revealed: Hb. 13·5 g.%; VPC 48%; WBC 10,000 per c.mm. with 76% polymorphs, 22% lymphocytes and 2% eosinophils. The erythrocyte sedimentation rate was 4 mm. in the first hour (Westergren). A blood smear was normal; there was no microspherocytosis.

Three further estimations of serum bilirubin were made over the next 5 days, with a negative van den Bergh reaction and levels of 1·4, 1·7 and 1·5 mg.%. The last 2 figures were obtained before and after 2 hours of exercise. The serum proteins were albumin 5·6 and globulin 2·7 g.%; alkaline phosphatase 3·5 King-Armstrong units; cholesterol 200 mg. A bromsulfalein test showed no retention of dye at 45 minutes. The prothrombin index was 100. Osmotic fragility of the red cells was normal. The reticulocyte count was 1·2%. Shumm's test for methaemalbuminaemia[5] was negative. The direct Coombs test was negative. An oral cholecystogram showed good function of the gall bladder; there were no calculi. Liver biopsy was essentially normal.

The patient's symptoms responded well to reassurance and the information that the tests performed had been normal.

DISCUSSION

On reviewing the data in this case it becomes apparent that there are no good reasons for confirming the original diagnosis of viral hepatitis. In retrospect it seems that he had chronic fatigue (see below) and a non-specific pyrexial

illness, at which time mild icterus was discovered. His urine had not been tested for bile or urobilin; the dark colour may well have been due to concentration associated with his fever. Only when that diagnosis was suggested did he develop symptoms of 'hepatitis.'

Throughout these studies there has been a preponderance of indirect-reacting bilirubin, the total figures now being not much lower than at the start of the 'illness', with a rather higher level just before admission. The investigations failed to reveal any evidence of liver dysfunction other than the hyperbilirubinaemia. It is noteworthy that severe exercise did not aggravate the clinical or biochemical situation; this also inclines the diagnosis against viral hepatitis—it will be remembered how, in the army, vigorous activity often led to a relapse.[4] The other studies completely excluded haemolysis as a cause of the jaundice.

The normal liver biopsy must be stressed. Smetana[11] considers that biopsy is the single most reliable laboratory test in the diagnosis of viral hepatitis. Six months after an acute attack one might not have expected degeneration and necrosis of liver cells, or periportal and intralobular cellular infiltration—although these might have been evident with continuing jaundice—but there should have been evidence of parenchymal regeneration. Smetana mentions that one lesion in particular persists for months in viral hepatitis—lipochrome pigment in the Kupffer cells, due to the phagocytosis of dead liver cells; this change was absent in the present case.

Watson[12] has stressed the importance of the one-minute direct-reacting bilirubin (1′)—corresponding to a prompt positive van den Bergh—in indicating actual liver disease or obstructive jaundice. His figures suggest that jaundice due to liver damage should be accompanied by a high 1′ level.

Recently Reichman and Davis[8] discussed their experience with American servicemen diagnosed as having suffered. from viral hepatitis. In particular they analyzed a number of cases in whom jaundice had apparently persisted after an attack, and have discovered that some of their patients had actually not had hepatitis at all. In 6 patients persistent elevation of the indirect fraction of the serum bilirubin was found. In only 1 of these was the diagnosis of hepatitis upheld—2 liver biopsies showed evidence of continuing inflammation. Another 2 patients were found to suffer from acholuric family jaundice, for which splenectomy was successfully performed; in neither of these had anaemia been significant. The remaining 3 cases had normal liver function tests and biopsies and fell into the group of constitutional hepatic dysfunction.

In 1902-1907 Gilbert and Lereboullet (quoted by Dameshek and Singer[2]) described cases of mild hyperbilirubinaemia in the absence of liver disease; it is now felt[2] that their familial cases were probably examples of mild haemolytic anaemia. In 1918 A. A. H. van den Bergh (quoted by Meulengracht[7]) stated that he had often encountered healthy people with 'physiological hyperbilirubinaemia.' Meulengracht[6, 7] collected 35 cases whom he labelled as having 'chronic intermittent juvenile jaundice' as most of his patients were diagnosed when aged 15-25 years. Apart from jaundice the only complaint was of periodic lassitude. In many cases he could trace hereditary occurrence, but this was not always obvious. Certain factors were found to precipitate lassitude and exacerbations of jaundice—'alcohol, convivial evenings, lack of sleep, sorrow and anxiety' and gastro-intestinal disturbances. The present patient complained first of fatigue, and he sometimes drank to excess; perhaps a 'convivial evening' had preceded his out-patient attendance at which the serum bilirubin was found to be 4·0 mg.%!

Dameshek and Singer[2] presented 3 families with this condition. They found no abnormalities other than an inability of the liver to handle bilirubin adequately; there was a greatly delayed excretion of injected bilirubin from the blood. There was no family history of jaundice in my patient, but his relatives live in Germany and communication with them has been scant.

It should be noted that the serum bilirubin level in constitutional hepatic dysfunction can be quite high—Sherlock[10] says it is rarely higher than 5 mg.%, but Watson[12] records a case with a 1′ level of 0·96 and a total of 8·8 mg.%.

Recent work by Cole and Lathe[1] and Rudi Schmid[9] has thrown much light on the chemistry of the bilirubins and the mechanism of the van den Bergh reaction—and, incidentally, on the nature of the defect in constitutional hepatic dysfunction. It has been shown that the indirect-reacting pigment is free crystalline bilirubin, which is insoluble. In the liver this is conjugated with glucuronide to form soluble direct-reacting bilirubin. *In vitro*, crystalline bilirubin can be rendered soluble by various means, as when alcohol is added in the second part of the van den Bergh test.

The implications of these facts in relation to constitutional hepatic dysfunction were discussed by Schmid at a meeting at the Massachusetts General Hospital in December 1956. He feels that the probable pathogenesis of this condition is the absence, on an hereditary basis, of the enzyme responsible for glucuronide conjugation of free bilirubin. He thinks that this is the heterozygous expression of the deficiency; the homozygous condition may be the rare 'congenital non-haemolytic jaundice with kernicterus' of infants. The features of this latter illness are as follows: High serum bilirubin (15-30 mg.%—all free bilirubin—but clinically they do not appear to be grossly jaundiced); no bilirubin in the bile, which is a pale yellow fluid; normally-coloured stools; death within the first year of life in most cases, after the development of severe basal ganglia signs, which are due to the deposition of insoluble free bilirubin.

This 'Gilbert' type of constitutional hepatic dysfunction is not the only form of constitutional hyperbilirubinaemia;

TABLE I. DISTINCTION BETWEEN 'GILBERT' AND 'DUBIN-JOHNSON' HYPERBILIRUBINAEMIA

	'Gilbert'	*'Dubin-Johnson'*
Urine colour	Light	Dark
Van den Bergh reaction	Indirect	Direct
Thymol and zinc turbidity	Normal	Abnormal
Bromsulfalein retention	Normal	Abnormal
Oral cholecystography ..	Good function	No excretion of dye
Pigment in liver cells ..	Absent	Present

another variety has recently been described by Dubin and Johnson.[3] Its distinction from the Gilbert group is shown in Table I. It, too, has an excellent prognosis. An important feature is the presence of an abnormal lipochrome pigment in the parenchymal cells of the liver.

SUMMARY

A case of constitutional hepatic dysfunction is presented, with a differential diagnosis of the causes of indirect-reacting hyperbilirubinaemia. It is of great importance to distinguish this condition from viral hepatitis. Recent discoveries in the field of bilirubin chemistry, and their clinical significance, are discussed.

I wish to thank Dr. Louis Mirvish for permission to report this case and for much useful advice about its presentation.

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TORN CONGENITAL DISCOID MENISCUS OF THE KNEE

CASE REPORT AND REVIEW

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Torn meniscus of the knee joint in young children appears to be a rare injury, even if an associated congenital anomaly of the cartilage is present. A case of congenital discoid cartilage presenting as an 'internal derangement of the knee', and proving at operation to be a torn cartilage is described.

CASE HISTORY

A female child R.V. aged 6 years and 9 months, was seen at the orthopaedic outpatients department on 16 April 1957. Six days previously, whilst playing, she was pushed and fell against a large stone, twisting her knee as she fell. The left knee became swollen and, although this had now almost subsided, it was still painful. Because of the pain she was unable to walk or extend the leg fully. Nothing of note in the previous history.

Pulse rate 90 per minute. Temperature 98·4°F. No abnormalities detected on general examination.

The right knee was normal on examination. The left knee was slightly swollen, there was no erythema; skin temperatures were equal on the two legs. The knee was held in 10° flexion; flexion-extension movements caused pain at the lateral aspect of the knee joint; there was a block to full extension. Movement was painful and only possible between 170°-90°. A tense, slightly tender swelling was palpable on the joint line antero-laterally, about half an inch in diameter, and this was not attached to the skin.

A diagnosis of internal derangement of the knee, possibly with a cyst of the lateral meniscus, was made and the child was admitted to the ward.

The patient did not improve on skin traction and on 29 April arthrotomy of the knee joint under general anaesthesia and a bloodless field was performed through a lateral vertical incision between the ilio-tibial tract and the patellar tendon with the knee in 90° flexion. The interior of the knee revealed a thickened disc-shaped, lateral cartilage with a transverse tear in the middle of the central portion and the meniscus was removed *in toto*. The post-operative period was uneventful and the child was discharged on 11 May 1957.

DISCUSSION

In view of the rarity of internal derangements of the knee in children of this age a review of the literature is presented.

Although discoid cartilages of the knee had been noted previously in anatomical dissections, the first clinical case was described by F. Kroiss in 1910. It was associated with a tear of the pathological cartilage. Since then 50 cases have been described. A list of such cases appears below, showing those with a definite history of trauma:

1910..	F. Kroiss	1 case with superadded tear.
1928..	A. Schulz	2 cases (1 traumatic).
1934..	J. G. Finder	3 cases (1 traumatic).
1935..	E. Bell-Jones	14 cases (8 traumatic).
1935..	W. Jaroschy	3 cases (1 traumatic).
1936..	A. G. Timbrell-Fisher	2 cases (1 traumatic).
1936..	D. Stewart Middleton	4 cases (1 traumatic).
1945 and previously	E. Cave and O. Staples	7 cases (6 traumatic).
1948..	I. S. Smillie	29 cases.
1957..	E. B. Kaplan	6 cases.

Incidence

Of all meniscal injuries reported, only a small fraction is associated with intrinsic abnormality of the cartilage itself. Cave and Staples in a series of 164 cases requiring meniscectomy described only 4 as having the congenital discoid pathology; less than half of such cases in the literature were associated with trauma. In the series of 1,300 cases of menisectomy reviewed by Smillie only 29 congenital discoid menisci were found.

Age and Sex

The writers on all series of cases requiring operation for damaged menisci agree that the tear is seen most commonly in active sporting enthusiasts between the ages of 20 and 30. In his review of 256 cases Melville Henderson tabulated the age incidence as follows:

Age in Years	*No. of Patients*
10–19	27
20–29	116
30–39	66
40–49	37
50–59	9
60–69	1

MacAusland in 1931 stated that the average age at operation in 388 cases was 21 years. In the cases associated with a discoid abnormality the age was younger. In the whole series only 2 cases presented under the age of 10 years. Case and Staples in 1941, reviewing 7 cases of discoid menisci, found the average age at operation to be 14 years. The present case appears to be one of the youngest recorded.

Recent reports on meniscectomy for ruptured meniscus agree that the ratio of female cases to male is 1 to 3 or 4. Of the cases associated with the defect under discussion, the series is too small for adequate assessment, but the numbers appear to be about equal.

Anatomy and Etiology

No report of medial discoid cartilages occurs in the literature. Two cases presented with bilateral abnormality of the lateral menisci.

Jaroschy, in his studies of comparative anatomy, expressed the belief that the highest evolutionary level of life in which a discoid cartilage occurred normally was the lizard. Recently Kaplan, after dissecting animals representing species of amphibians, reptiles, birds and mammals, found no disc-shaped cartilages in any of these life forms; certain of them, however, normally exhibited a circular meniscus. He has also investigated the embryology of the normal cartilage in man and concludes that at no time from the earliest days of development does a disc-shaped meniscus occur in the foetus. The semilunar shape of cartilage is laid down early in the mesenchymal plate, interposed between tibia and femur. Observations made at the removal of discoid menisci show that no attachment of the posterior horn to the tibial platform exists. Instead, a continuous menisco-femoral ligament (ligament of Wrisberg) links the posterior horn of the meniscus with the lateral side of the medial condyle of the femur. This is similar to the arrangement in animals other than man. He concludes that the pathological condition now under discussion represents the final dynamic state of a congenital anomaly of the attachment of the posterior horn of the meniscus, the discoid shape being a hypertrophy of a semilunar cartilage caused by abnormal movements allowed by the defect.

This view is contrary to the following widely held theory of the etiology of the condition: It is believed that, when cartilage is deposited in the mesenchymal plate which is interposed between the tibia and femur in the foetus, it does so in a disc shape. Normally this shape is modified into that of the adult semilunar cartilage. It is believed that in the condition under discussion this re-absorption does not take place.

Clinical Presentation

Cases of discoid meniscus have presented in two clinical ways, viz. with or without trauma. The trauma may be severe or minimal.

1. *Without Trauma.* The outstanding complaint is a snapping sensation in the knee, accompanied by a jar, thud or click, when the knee is extended. It is sometimes associated with pain and episodic effusions into the joint. Movements are frequently unrestricted. Occasionally a tender mass is palpable along the joint line.

2. *With Trauma.* An internal derangement of the knee following injury is seen at a younger average age than the usual Torn Cartilage case. It is in this category that the present case falls.

Anatomical Dissection. Reports of discovery of the condition on dissection are found, in the literature, but no figures of the incidence are given.

Treatment

All cases in the literature were proved by meniscectomy and it is agreed that the operation cures the condition.

CONCLUSION

The condition of discoid lateral cartilage of the knee, whatever its mode of presentation, is an unusual entity. It is even less common when occurring in association with a super-added tear.

A review of the literature on comparative anatomy of the knee cartilages throws doubt on the presence of the discoid shape in animal life other than man. Doubt is further thrown on the theory that this condition in man is of congenital etiology; a theory is discussed which suggests that the anomaly is one affecting the attachment of the posterior horn, the discoid shape being a secondary hypertrophy following abnormal mechanics and stresses on the cartilage.

SUMMARY

A case is presented of internal derangement of the knee in a female child 6 years and 9 months old. Operation proved the case to be one of torn lateral cartilage, with a congenital discoid anomaly as the underlying pathology. Meniscectomy was successfully performed.

The case presented appears to be of interest because of the very young age of the child and the rarity of the condition described. Although the literature records few cases of discoid lateral menisci, most orthopaedic surgeons agree that the defect itself is not such a rare abnormality; it is the association of the condition with a tear of the cartilage that is unusual.

A review of the literature and discussion of the condition is presented.

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SOME AFRICAN CARDIOPATHIES

REPORT OF A JOINT SEMINAR OF THE DEPARTMENTS OF PATHOLOGY AND MEDICINE OF THE UNIVERSITY OF THE WITWATERSRAND

Dr. M. McGregor: The object of our seminar this morning is to ascertain the degree of identity if any between 3 unusual forms of heart disease which have been described in Africa. The condition now known as 'Endomyocardial Fibrosis' (EMF) was first associated with the African when Bedford and Konstam[1] reported its presence in African troops at the end of World War II. In 1948 Prof. Davies published his first reports of this condition in Uganda.[2, 3] This and subsequent publications by Davies, Ball and Williams[4, 5] made it clear that it was an extremely common condition at least in this part of Africa. In Johannesburg in 1951 Gillanders[6] and in 1952 Higginson, Gillanders and Murray[7] described an African cardiopathy in the Bantu population around Johannesburg which appeared to have sufficient features in common with endomyocardial fibrosis to suggest to Davies and Ball[8] that it might represent an early severe form of the condition described by them. However, in many respects it appeared to be very dissimilar. In 1953 Becker, Chatgidakis and van Lingen described in Johannesburg yet another cardiopathy[9] which appeared to have some features in common with the other two conditions. Unlike them, however, it occurred in both White and African subjects. To those of us who are not pathologists, and possibly even to some who are, it is a confusing picture. Do these three conditions overlap, and if so to what extent? We are very fortunate in having one of the authors of each of these descriptions here today. We shall ask them, after viewing each other's material, to attempt to answer these questions for us.

I will ask Prof. Davies first of all to describe the characteristics of *Endomyocardial Fibrosis* in Uganda and to stress the similarities and differences between that condition and the two cardiopathies described in Johannesburg.

ADDRESS BY PROF. J. N. P. DAVIES

In Uganda, out of 100 cases of heart failure about 30% cannot be placed in a conventional clinical diagnostic category. This is in a population in whom certain diseases, such as thyrotoxicotic heart disease and coronary thrombosis are almost totally absent. Rheumatic heart disease, renal hypertension, aortic syphilis, bacterial endocarditis, tuberculous or purulent pericarditis make up many of the remainder. But there is still this large category of clinically unexplained heart failure. At post-mortem examination of patients dead of heart disease we find, as do the clinicians, that there are still about 30% which will not fit into a conventional pathological diagnosis. Of these about 15% turn out to be EMF, and of the rest another 5-7% are forms of true myocarditis, of which there appear to be several varieties. There is an eosinophilic myocarditis, a myocarditis of Fiedler's type, both localized and diffuse, and there is what we assume to be virus myocarditis of varying degrees of intensity. There is about another 5% of cares of unexplained myocardial failure with mural thrombus or endocardial thickening; some of these are hypertrophied. Yet another 5% will show hearts with mural thrombosis, but associated with cirrhosis of the liver or other forms of liver disease. These hearts are often small and the heart disease has often been clinically quite overshadowed by the liver disease. About 15% of all heart disease in Uganda is due to EMF, and it is this group I wish to discuss today, not forgetting that it is not the only unusual type of heart disease in Uganda.

I would say the dominant features of this type of heart disease are as follows:

1. Enormous fibrosis giving a thick rugose appearance of the endocardium, like sugar icing.
2. Both ventricles are affected and not infrequently the atria.
3. The fibrosis tends to have a sharp rolled-over edge.
4. There is sometimes a mass of clot over the surface of this layer of fibrous tissue. This is enormously thick in contrast with the cases of Prof. Becker which I have seen, and this clot is superimposed on a background of very thick and old fibrous tissue.
5. There is involvement of the papillary muscles, chordae and cusps in this fibrous tissue.
6. The area of involvement is the inflow tract of the ventricles running up to behind the A.V. valves, the posterior cusps of which sometimes completely disappear, so that from the atrium right down to the ventricles and down to the apex there is a thick sheet of fibrous tissue as much as a centimetre thick. In shape therefore the fibrous area resembles one of those old-fashioned candle snuffers, inverted, with the cone below and a long handle.
7. The semilunar valves, the aorta and pulmonary valves and the pulmonary artery are never affected.
8. The right ventricle, when it is severely affected, shows the most extraordinary lesion, which you can clearly recognize from the outside by a depressed area over the right apex. This is due to the complete obliteration of the apex cavity.
9. We never see emboli in any of our cases, old or recent, save where there is an acute terminal bacterial endocarditis superimposed. I base the statement of the rarity of the embolism on an experience of over 100 cases.
10. Very considerable calcification may be present, either in the mural endocardium or in the valves.
11. Histologically the most characteristic lesion in the myocardial fibres is a watery vacuolation. No major vascular lesions are seen. There is little or no elastosis.

In conclusion, I find that the lesions Prof. Becker has shown to me in his material cannot be paralleled in the EMF material in Uganda. We have not studied our cases of unexplained heart failure with the care that he has given to his cases, but my impression is still that I have not seen in those I have looked at in Uganda the lesions that Prof. Becker has demonstrated to me here. It seems to me that Prof. Becker's material is of a totally different type to anything we have noted in Uganda.

Dr. McGregor: Dr. Higginson will you now describe for us the salient features of the condition which you and Dr. Gillanders described under the name of *Nutritional Heart Disease*?

ADDRESS BY DR. J. HIGGINSON

My material is derived from the African (Bantu) Hospital at Baragwanath where Dr. A. Gillanders was a member of the staff when I first went there. My first post-mortem examination from him was on a man with normotensive heart failure. All I could find at autopsy, apart from passive congestion, was a big heart and I was at a loss to find the cause.

It was then I had the opportunity of reading Dr. Gillanders' work. He had demonstrated that certain cases of heart failure admitted to Baragwanath Hospital were of the low-output type. In hospital they would get better on a good ward diet, go home, return after a few weeks to hospital in failure again, and again recover and go home. Eventually they would die in intractable failure and come to me in the autopsy room. Dr. Gillanders claimed that he could return some of these cases to failure in hospital by feeding a poor diet based on that eaten at home. As far as I know, no one else has repeated this experiment. More recently, Dr. H. Grusin has further classified these unusual cardiac cases clinically. He has divided them into 3 groups; a group very similar to that described by Gillanders; a group that apparently is of the beri-beri type; and a third group of uncertain nature with high output failure. In the post-mortem room the majority of cases we have seen have been of the type originally described by Gillanders. Of 360 cases of heart failure seen by me roughly 16% were essentially cases of big hearts without an apparent cause. These we classified as follows: (i) Cases with no more than a big heart, (ii) cases with a big heart and intracardiac clot, organized to varying degree and (iii) cases in which there was a big heart with some muscle damage.

The existence of this last group as a separate entity is somewhat doubtful, for in my experience one may find mild degrees of myocardial fibrosis in various conditions, such as hypertensive cardiac hypertrophy and chronic rheumatic heart failure. Further, endocardial thrombus often merges with myocardial fibrosis.

To summarize, the essential pathological pattern of atypical heart failure seen in Baragwanath Hospital is that of a big heart with or without intracavity thrombosis, sometimes associated with myocardial damage. In addition we have seen some hearts with quite severe myocardial damage, suggesting the late stages of a

myocarditis such as 'Loeffler's' or 'Fiedler's' myocarditis. I am excluding these latter from the discussion, because they are few and their relationship to the common type is doubtful.

The essential pathogenesis of these cases with intracardiac thrombi appears to me as follows:

The basic disorder in all types, including that which has been described as 'Nutritional heart disease', seems to be cardiac dilatation. This is followed by a deposition of thrombus on the endocardium, in some cases without obvious endocardial damage. There is no evidence of a fibrous ischaemia or other lesion of the myocardium in most cases. Frequently, however, in the apical region of these large dilated hearts there are isolated thrombi lying between the trabeculae, and in some cases we have seen areas of apical fibrosis here which are similar to that described by Prof. Becker. I believe, however, that these are due to organized thrombi. Only a very occasional case shows the heavy fibrosis of the endocardium such as that described by Prof. Davies. We have never yet seen valvular involvement.

Dr. McGregor: Prof. Becker would you now describe what you consider to be the salient features of the condition you have named *Cardio-vascular Collagenosis with Parietal Endocardial Thrombosis* and compare and contrast its features with those which the last two speakers have described?

ADDRESS BY PROF. B. J. P. BECKER

At first, in order to gain some sort of perspective, I would like to say that in our autopsy series idiopathic heart disease forms about 10% of all cases who have died from cardiac causes. Of these cases of idiopathic heart disease, the type which I and my co-authors have described as cardio-vascular collagenosis with parietal endocardial thrombosis form the great majority. In addition we see a few cases of idiopathic myocarditis of the Fiedler type; we see a few cases of idiopathic hypertrophy; and we see a few cases dying from acute or chronic cardiac failure in which even after exhaustive investigations we can find no cause.

In the condition which we have described, there are 3 major features: Firstly, there is evidence of congestive cardiac failure, secondly, most of them will show evidence of visceral infarction and, thirdly, they have a characteristic cardiac lesion. I do not wish to say much about the clinical side of the picture, except that congestive failure may be of a rapidly progressive type with an average duration of about 6 months and with a range of less than 1 month to 3 years before death. At autopsy all the characteristic signs of congestive cardiac failure are present. A striking feature, however, is the finding of solitary or multiple infarction of one or more viscera in over three-quarters of all the cases. Infarctions are always bland and may be both recent and old and we think that in the majority of cases the cause of these infarctions is clearly embolic, the source of the embolus being antemortem thrombus formation in either the right or left side of the heart. The third feature of these cases is the characteristic cardiac lesion. The heart is enlarged, and there is dilatation with or without hypertrophy; at any rate we feel that hypertrophy is not a necessary part of the condition. Mural thrombosis may occur in any of the cardiac cavities but is most commonly found in our experiences in the left ventricle. Underlying the mural thrombosis there is an area of sub-endocardial necrosis or in some cases sub-endocardial fibrosis. There is an absence of significant lesions in the pericardium, the cardiac valves, the coronary vessels and in the aorta.

We had examined many of these cases and had observed nothing aetiologically revealing, and made little or no progress until one day in the autopsy room we examined a young adult who had died of a fulminating congestive cardiac failure. The heart was enlarged and dilated, there was no evidence of any mural thrombus in the heart, but on very close examination we were intrigued to see a very fine film of fibrin on the endocardial surface. The examination of the endocardium in this case gave us an indication of what we consider to be the early primary lesion of the endocardium.

The endocardium of the normal heart is an inconspicuous structure and when you stain it with a dye such as toluidine blue there is normally no metachromatic reaction. In early cases of this condition we have observed a swelling of the endocardium which is accompanied by a metachromatic reaction. There is an excess of a characteristic muco-polysaccharide which justifies the diagnosis of a true acute mucoid oedema. The metachromasia is red-violet, it is alcohol resistant and it persists after treatment with both testicular and streptococcal hyaluronidase. It is, we feel, a specific muco-polysaccharide with reactions suggesting chondroitin sulphate B, and we have not found evidence of this reaction in a series of normal control hearts which we have examined. It is well known that an acute mucoid oedema is accompanied by profound local changes, such as increase in permeability, a transmineralization, and an intense local anoxia. We have surmised in these cases that if the first reaction is, as we believe, an acute mucoid oedema, then as a result of the consequent marked increase in permeability, fibrin exudes onto the surface of the endocardium and into the tissues of the endocardium. This fibrinous exudation of the surface might be the forerunner of the mural thrombus which would build up in the areas in which mucoid oedema has occurred. Following the intense tissue anoxia which may result from the mucoid oedema, sub-endocardial necrosis could theoretically occur.

The sub-endocardial myocardium receives most of its blood supply from the cavity of the heart. Once the thrombus has built up on the surface, local anoxia would become more marked, and this is a possible way in which the sub-endocardial necrosis could develop. A striking feature of this mucoid oedema is that it may be strictly focal, and we have observed these lesions in the form of endocardial polypi consisting of mucoid oedema infiltrated with fibrin and in some cases showing fibrinoid necrosis.

We have observed also additional pathological features of considerable interest. There is interstitial myocardial oedema causing separation of muscle bundles, a type of serous myocarditis, and muscle degeneration occurring chiefly in the inner third of the myocardium. The degeneration consists of hydropic degeneration, necrobiosis, granular disintegration of fibres, and foci of basophilic degeneration. In the Thebesian and luminal vessels, and in the myocardial venules and arterioles, curious lesions also occur. These are curious papillary lesions consisting of oedematous connective tissue, sometimes infiltrated with fibrin, reminiscent of the arteritis verrucosa which has been described by Holsty. Some of these papillary lesions in the blood vessels are very narrow-based and may be a possible source of some of the minuter microscopic emboli which we have observed in the organs, particularly in the glomeruli. In addition there is fibrinoid necrosis. Fibrinoid necrosis is a difficult lesion to prove adequately. At the time when we wrote our article[9] we believed that we were dealing with a true fibrinoid necrosis and not a fibrinous infiltration. Nowadays there is a tendency to believe that fibrinoid itself represents fibrin which has infiltrated the tissues and has become altered by the local conditions existing in the tissues. However, foci of fibrinoid have been observed focally and sometimes diffusely in the smaller vessels of the heart.

Briefly then we believe that the 'target organ' here is the muco-polysaccharide of the mural endocardium. The first reaction is a mucoid oedema which may either resolve completely or may, if it is severe enough, lead by increased permeability to fibrinous exudation onto the surface and into the tissues. Secondly, as a result of the anoxia, sub-endocardial necrosis may ensue. There will then be a building up of mural thrombus, which will undergo organization, this being the stage which we have designated subacute; that is, the picture of organizing thrombus on the mural endocardium overlying an area of endocardial necrosis in which the necrotic tissue is being replaced by granulation tissue. The final stage consists of fibro-elastosis which is frequently focal and which results in the great majority of cases from the organization of the necrotic endocardium and the overlying thrombus. We believe, however, purely on theoretical grounds, that it is not necessary in every case for the acute mucoid oedema to proceed to overlying thrombus and necrosis, but that it may, by a process of fibrillogenesis, proceed directly to a fibro-elastosis. The fibro-elastosis which we have observed in our cases is never very severe. It consists of focal endocardial plaques, either at the apex of the heart overlying the papillary muscles, in the auricles, and on the outflow tract of the heart in the upper part of the interventricular septum. I have not observed, except very rarely indeed, the gross degree of endocardial sclerosis and fibrosis which Prof. Davies has demonstrated to us here today.

There is another occasional interesting lesion found involving the intima of the pulmonary vessels, consisting of a giant-cell intimitis, and one has wondered whether or not a similar change in the intima is not perhaps affecting the hepatic veins because the liver in these cases shows a very acute central congestion approximating to a Chiari syndrome.

As regards the aetiology, for the sake of brevity, we did not publish our views in our original publication. I do not think that our cases are due to malnutrition. We have observed this condition in all social levels of the population, in Europeans and Bantu, and in most of our European cases and in some of our non-European cases no evidence of malnutrition whatsoever has been observed. It *may* be some very unusual type of deficiency condition, but I personally do not feel that that is the case. We have attempted in the cases observed over the last 2 years or so to find a virus, and portions of heart muscle have been sent to the virologists, with so far negative results. Following the recent reports in England that this type of case may be due in some way to toxoplasmosis, in the last half-dozen cases which we have observed we have attempted to isolate toxoplasma (with the aid of experts of the South African Institute for Medical Research) but so far we have had negative results. Finally, one has attempted to reproduce the condition experimentally on the basis of hypersensitivity but without success as yet.

DISCUSSION

Dr. McGregor: I think it is clear from what has been said so far that Prof. Becker and Prof. Davies feel quite clearly that the conditions which they have found in Johannesburg and in Kampala respectively, do not overlap at all.

Prof. Davies could you tell us if you are in a position to say, to what extent, if at all, the condition you have seen at Kampala and that described by Dr. Higginson coincide?

Prof. Davies: There seems to me to be very considerable difference. We see in Uganda a large number of a very distinct and gross form of heart disease, different from what has been described in Johannesburg. There is, however, a great variety of conditions which we can recognize as precursor states. If the conditions described by Dr. Higginson and Prof. Becker are in fact early stages of the condition which we in Uganda recognize as EMF with its advanced fibrosis, why are the early stages so uncommon in Uganda? Why are the late stages so uncommon in Johannesburg? Both clinically and pathologically it seems to me that both conditions described in this city differ markedly from EMF. In particular, there is the great rarity of embolic phenomena in EMF. I understand in Dr. Higginson's cases, as well as those of Prof. Becker, embolic phenomena are common and important. At one time we wondered if the absence of embolic phenomena in EMF might be explained on the ground that the heart's action was so weak that clots would not be forced off the endocardium. I cannot believe that this is the explanation, just as I do not believe that ventricular stasis is an aetiological factor in EMF. It seems to me that we are discussing two and possibly three different types of heart pathology here this afternoon. The lesions that Prof. Becker has described are different from anything we have seen in Uganda which we have defined as EMF. It is possible that we do have some cases similar to those of Prof. Becker in our unexplained heart cases. I was at one time impressed with the evidence in some Uganda cases of endocardial and myocardial necrosis, and though I could trace a connection between such degenerative lesions and EMF, but in recent years I have been less impressed and remain quite uncertain whether there is any connection between the conditions.

No one has been able to suggest any satisfactory aetiological cause of EMF. We have been unable to incriminate any parasite. Serological tests against certain viruses are negative. While we cannot exclude a malnutritional cause there is little evidence to suggest that these patients are particularly malnourished, nor as far as I am aware is there anything in the world literature dealing with malnutrition which corresponds to the sort of lesions we see in EMF, though of course endocardial mural thrombi are reported.

Swiss colleagues whose opinions I have sought have stated that the lesions seen in EMF appear to be different from those they are familiar with in cases of Loeffler's syndrome.

There is, however, widely distributed over the world, a series of descriptions of a form of heart disease in which lesions similar to those of EMF are described. Such descriptions emanate from many parts of Africa and from Europe and North America. Perhaps it particularly affects those who have been in the tropics, but there are certainly some cases in people who have never visited the tropics. Aetiologically speaking we have not a clue. Pathologically we are seeing only the end stages. The nature of the intermediate and early stages, as far as I can see, are hypothetical.

It appears to me that we can conclude that there are at least two common heart diseases of unexplained aetiology in Africa, possibly three, and perhaps more. It seems to me that Prof. Becker has put up a very convincing case for a form of heart disease whose lesions appear to be quite specific and different from anything I have seen elsewhere, including Uganda, except possibly in very rare instances. The frequency with which such cases appear here is remarkable. Dr. Higginson's cases seem to me to differ in certain respects from those of Prof. Becker.

There is one point I would like to stress. Prof. Becker drew attention to the marked elastosis, which he regards as a terminal stage, and to the sharp demarcation of the myocardium from the thickened elastotic endocardium in his cases. These two points are sharply at variance with what we see in EMF. We do not see elastosis except for a small isolated irregular patch quite different from the lesions he has demonstrated in his late stages. We do see occasional endocardial fibro-elastosis in African children in Uganda and we have seen cases of what we took to be endocardial fibro-elastosis in adults. Perhaps these few cases are related to the condition described by Prof. Becker. At any rate we recognized their difference from EMF by the fact that the endocardial thickening affected the outflow tract and particularly the area below the aortic valves, in contrast to EMF, where the lesions are in the inflow tract areas. The restriction of the EMF lesions to the inflow tract areas is very striking and may provide an aetiological clue. By contrast, the lesions in Prof. Becker's cases seem to be much more diffuse with no particular tendency to localize in the inflow tract. In brief, I have seen nothing here today which suggests to my mind that there is any relationship between EMF and the forms of heart disease described from Johannesburg.

Dr. McGregor: To summarize very briefly, it would seem that we are all probably going to agree that EMF is a different disease to that commonly seen in Johannesburg. Dr. Higginson and Prof. Becker have, it appears, both very, very rarely seen a case of typical EMF as described by Prof. Davies and each of them has recognized it as a disease entirely different to the ones they have described and published from Johannesburg. Prof. Davies has, very rarely, seen cases similar to those seen in Johannesburg. All three speakers have seen and recognized fibroelastosis and Loeffler's and Fiedler's myocarditis as separate entities.

The main differences then are that EMF is a most extensive fibrotic lesion with well-defined edges. By contrast, in the other two lesions fibrosis is absent or slight and seems to fade off into normal tissue. The fibrosis in EMF runs deeply into the myocardium, typically 5-10 mm. in depth. By contrast, in both the local conditions where fibrosis is evident it is an extremely superficial feature. Next, EMF involves particularly the apex and the inflow of the ventricles, and because of this there is frequent involvement of the mitral and tricuspid valves and never involvement of the aortic and pulmonary valves.

Prof. Davies: May I make an interjection here. There is never any evidence, even in the involved valves, of valvulitis of the valve itself. This makes it distinctive in my opinion from both rheumatic valvulitis and Loeffler's disease.

Dr. McGregor: Thank you Prof. Davies. By contrast with EMF, in neither of the local conditions have valve lesions been demonstrated, except the secondary valve involvement which rarely occurs when the thrombus builds up sufficiently by extension to bind down the papillary muscles and chordae tendineae. Next, Prof. Davies has never seen the appearance of elastosis in EMF which we saw demonstrated so beautifully in some of Prof. Becker's slides. Dr. Higginson also finds elastosis comparable to that shown by Prof. Becker. Next, although all three conditions frequently have antemortem thrombus in the ventricle, in 100 cases Prof. Davies has never seen evidence of systemic embolization. Both Prof. Becker and Dr. Higginson, however, frequently do see it. I think these are the major differences that have been apparent to me during this discussion.

I would now like to go on to enquire whether we may differentiate the two Johannesburg diseases. Dr. Higginson, would you like to comment on whether you think there is identity or difference between the pathology you have described at Baragwanath and that described by Prof. Becker?

Dr. Higginson: The difference between th type of heart I have been describing and the condition described by Prof. Becker is partly due to a difference in interpretation. I do think that there

is probably a large degree of agreement that the final pictures which we have seen are similar in appearance. The differences that do exist seem to me to be a matter of interpretation of these appearances.

Dr. McGregor: Prof. Becker would you agree with Dr. Higginson's opinion that most of his cases appear pathologically to overlap with your own.

Prof. Becker: Yes, I feel the cases which we have been seeing at Baragwanath, and cases which we see here in Johannesburg, are essentially the same condition. I think, as Dr. Higginson has said, that we differ only on pathogenesis and interpretation. Dr. Higginson thinks that the thrombus comes first and that the aetiology of the thrombus may lie in some metabolic disorder. I feel that there is some local cause. He is concentrating on a different feature of the Virchow triad here.

Dr. McGregor: In spite, however, of obvious similarities in pathological appearance there are equally obvious differences at a clinical level. Dr. Gillanders described a form of heart failure which was at first amenable to good dietary treatment and could be reversed, and which later on became chronic and went progressively downhill. Dr. van Lingen,[9] on the other hand, in describing the clinical features of 'cardiac collagenosis' left the impression of a rapidly downhill course without any response to any form of treatment. Do you feel, Dr. van Lingen, that these differences are possibly not as great as first appeared? Have you with the passage of time come across cases with a less acutely downhill course, cases which correspond more closely to the clinical condition described by Dr. Gillanders?

Dr. B. van Lingen: The surprising thing about the clinical side of this study was the way in which one could divide the cases on clinical grounds into acute, subacute and chronic, and the way in which Prof. Becker was able to find a pathological appearance which appeared to correspond with the clinical picture. A case which on clinical grounds was acute, that is, which had a course of 6 months or less, appeared to have a predominance of necrotic lesions, while a case which had a chronic downhill course, lasting in some cases up to 3 years, would show predominant fibrosis and elastosis. Since we published our work[9] I have seen several cases, subsequently proved pathologically, in whom the clinically recognized course has lasted much longer than 3 years. There is still, however, the acute group characterized by bouts of pyrexia, sometimes leucocytosis, multiple thrombotic episodes, relapses into acute congestive cardiac failure, and a rapid downhill course. So far as I am aware this acute course has not been reported from Baragwanath Hospital amongst the African patients.

Dr. McGregor: It seems that there is a greater overlap in the clinical appearances of these two conditions than was apparent some years ago when they were first described. You, Dr. van Lingen, now recognize more chronic cases which appear in no way different clinically from the syndrome described by Dr. Gillanders. Dr. Keeley, you who are working at Baragwanath presumably still see the condition originally described by Dr. Gillanders. Could you tell us whether you feel that there are in fact some acute cases which possibly correspond to the acute cases reported by Dr. van Lingen and his colleagues?

Dr. K. J. Keeley: One does see large numbers of cases of heart failure for which one can find no explanation. This heart failure of unknown aetiology is in fact the commonest diagnosis made, ranking higher than rheumatic and hypertensive heart disease. For the most part the course of the disease is still very much as was originally described by Dr. Gillanders, that is, one of repeated admissions for heart failure which at first responds to hospital treatment and later becomes resistant. Occasionally, however, cases do come in and run an extremely short course. There is also a small sub-group of women whose failure has been precipitated in the postpartum period.

Dr. McGregor: To summarize, I take it then that you still see the typical chronic downhill course described by Dr. Gillanders, but that more recently you have recognized some cases of a very acute nature similar to those described by Dr. van Lingen.

CONCLUSION

Dr. McGregor: I feel that I have learned a great deal this morning which I did not know before. I have learned that so-called *Nutritional Heart Disease* described in Johannesburg Africans is almost certainly the same condition as *Cardio-vascular Collagenosis with Parietal Endocardial Thrombosis* occurring in both European and African races in Johannesburg. I have also learned that both these conditions differ markedly from *Endomyocardial Fibrosis* in Uganda and if there is in fact some common aetiological factor it is as yet unknown. EMF, in short, could not be bracketed with these conditions. Finally, I have been reassured that none of these conditions appear similar to congenital cardiac fibroelastosis, or to the pathologies described by Loeffler or Fiedler.

The naming of these three African cardiopathies is perhaps unfortunate and may partially be responsible for the confusion which has existed up to now. One can take no exception to *Endomyocardial Fibrosis*, which is purely a descriptive term. The term *Nutritional Heart Disease* implies, however, an aetiology which is by no means proved and to some appears improbable. The term *Collagenosis* is perfectly valid in the sense in which Prof. Becker has used it but carries the unfortunate connotation in medical minds of a system disease. One even wonders whether we could not persuade Prof. Becker and Dr. Higgins to adopt a more non-committal eponym until a more descriptive or enlightening name can be found.

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SOUTH AFRICAN MEDICAL CONGRESS, DURBAN SCIENTIFIC EXHIBITION

This Exhibition is to be held in the Old St. John's Ambulance Hall at Cartwright's Flats, which is a large hall which will adequately house all the exhibits. There are approximately 50 exhibitors and there is to be a historical section and a purely scientific section. The exhibitors are from all the principal Government Departments and the Medical Schools of Cape Town, Johannesburg and Durban.

There are exhibitors from overseas. The Welcome Research Museum is sending out a large exhibit and the Director of this museum is attending in person. There are also exhibits from the Plastic Surgery Department, Odstock Hospital, Salisbury, England, and the Cornell Medical School, New York, as well as from Northern and Southern Rhodesia.

The Exhibition will be open all day for members of Congress and for the public in the evenings, and it is proposed, should the public patronize it sufficiently, to extend the duration of the Exhibition for a week beyond Congress for them.

This Exhibition promises to be one of the best yet held. An Information Bureau will be provided and the Old St. John's Hall will be in telephonic contact with the main centre of Congress activities. Arrangements are being made to run a shuttle bus-service between the main Congress centre and the old St. John's Hall, which is a comparatively short distance. The exhibition will be opened on Monday, 16 September at 11 a.m. by Prof. E. G. Malherbe, Principal of the Natal University, when his Worship the Mayor of Durban (Councillor Percy Osborn) and the visiting guest speakers from overseas will be present.

MEDICAL AID SERVICES IN SOUTHERN RHODESIA

ALDERMAN NIGEL PHILIP, O.B.E.

Salisbury, Southern Rhodesia

Insurance of property and goods is effected because any loss might seriously affect the financial position of the owner. The fact that facilities are always easily available and that premiums are reasonable also has considerable influence. In health insurance, however, the risk of financial embarrassment is not so apparent; or, rather, the person concerned is always hopeful that with reasonable care good health will continue indefinitely. That this hope is often shattered has been the experience of the Rhodesian State Lottery Trustees in dealing with cases of distress caused by illness and, as their funds do not permit of a general acceptance of applications for assistance, it was felt that something had to be done to solve a very pressing problem. One of the Trustees undertook to find a solution.

Having had some experience in the administration of medical aid societies and having myself greatly benefited through such societies, I had little doubt that the answer to the problem was the expansion of medical aid societies; but how was this to be done? This is what happened:

RHODESIAN ORIGIN

In February 1939, a resolution was submitted to the Rotary Club of Salisbury advocating (*a*) the formation of an association of existing medical aid societies, and (*b*) the expansion of medical aid societies generally by the combined efforts of all employers. In regard to (*b*) it was felt that expansion would be difficult unless some scheme was formulated whereby all employers, even those with only one or two employees, could participate. This scheme was something entirely new and original in its conception and can be claimed as of Rhodesian origin, no similar organization of this kind having been known at this date.

Before these resolutions could be implemented, considerable propaganda was necessary. Moreover, the war years intervened, but eventually by the ready acceptance of the scheme by employers, encouraged by the Chamber of Commerce and the Chambers of Industry throughout the country, there came into being the Representative Council of Medical Aid Societies of Rhodesia, the Commercial and Industrial Medical Aid Society, and the Matabeleland Medical Aid Society. A Society formed on similar lines has been started at Lusaka.

The Commercial and Industrial Medical Aid Society was the first to be formed and started operations on 1 October 1945. Membership was open to all members of Chambers of Commerce and Chambers of Industry throughout Southern Rhodesia. Later, however, Matabeleland representatives on the management committee decided, after joining and gaining the necessary knowledge, to secede and form their own society, operated under a similar constitution. Every facility was afforded them and Bulawayo members were transferred to this new society. This society was started in about 1947 and is known as the Matabeleland Medical Aid Society.

Membership of these societies was first confined to all firms and their employees, including Coloured, who were members of the Chambers of Commerce and Chambers of Industry, but applications from other employers became so insistent that membership was made available to all reputable firms and other bodies.

SELECT COMMITTEE

As these societies grew in strength and the benefits became more and more evident, there was a feeling that membership should also be made available to the rural population—farmers, miners, etc., and in 1953 a Parliamentary Select Committee was appointed with Mr. Ian Smith, M.P., who had strongly advocated such a move, as chairman. In accepting the motion the Minister of Health said: 'The Government welcomes the interest the House is taking in this world-wide problem of the cost of Public Health services, and the Government accepts the motion. If an all-party Select Committee of this House can succeed in devising a scheme under which it can bring Public Health services to the population of this country *without increasing the subsidy which would have to come out of taxation*, such a Committee would deserve the thanks of the country'.

The Select Committee's report contained the following paragraphs: 'Our next problem was to consider how we could initiate such a scheme. The most obvious method to us seemed to be to try to encourage one of the present schemes to expand and cover the country districts. Now, I think the House will appreciate that there are difficulties attached to a suggestion such as this. We had considerable help in particular from one of the biggest schemes in the country, the Commercial and Industrial Medical Aid Society of Southern Rhodesia. They gave us evidence and were willing to help us in every possible way, but they did point out the disadvantages and the dangers which would be attached to their throwing their doors open to a completely new block of people who were not of the kind that they already catered for'.

These quotations from *Hansard* have been made to indicate that the Government did appreciate the service to the country made by Medical Aid Societies, and that the Select Committee, after taking considerable evidence, was satisfied that the solution to their problem lay in the expansion of the Societies formed under the Group System such as the Commercial and Industrial Medical Aid Society.

GROUP SYSTEM

This Society has, in fact, now opened its doors to the rural population, but with some restrictions in regard to mileage charges in order to put these members on a similar basis to the urban dwellers. In addition, as confidence has been gained over the years, the management committee has accepted practically all sections of the population, and there is a steady increase in membership throughout the country. The total number of people receiving the benefits of health insurance under the Group System today, excluding those insured under other types of medical aid societies, is approximately 35,000.

The Group System can be described as a joining of forces of all the employers in close cooperation with their employees, and the success which this system has enjoyed can be ascribed in full measure to the generous contribution by employers, whose support amounts to bearing half the medical expenses of their employees with wife and children included. As the system is capable of embracing the whole country, and there appears no limit to its expansion today, it can prove an alternative to a State medical scheme, and *without any cost to the general taxpayer*.

The cost of a State medical scheme has proved quite unpredictable, and the British scheme, estimated to cost £170 million, now costs £690 million. In 1945 a commission appointed by the Southern Rhodesian Government to report on the feasibility of a State medical scheme, and of which Prof. C. F. M. Saint was chairman, recommended three schemes for consideration costing respectively £350,000, £400,000 and £500,000 per annum. The cost today, with the great increase of population and the higher cost of every article and services generally, would be more than double, even if abuse could be controlled, which is doubtful. None of these schemes was ever implemented. It should be realized, however, that the Group System is not immune to a similar risk, and experience has already shown that its solvency can only be guaranteed by a strong and vigilant management committee, untrammelled by political influences and supported by a manager of the highest qualifications.

REPRESENTATIVE COUNCIL OF MEDICAL AID SOCIETIES OF SOUTHERN RHODESIA

The Salisbury Rotary Club's resolution (*a*), advocating the formation of an association of existing medical aid societies, was duly implemented. Three of the main objects were (*a*) pooling knowledge and experience of medical aid societies' practices; (*b*) assisting in maintaining uniformity of relationship between member societies and the medical profession; and (*c*) assisting and encouraging the formation of medical aid societies.

The Representative Council started in 1946 with foundation members consisting of 12 societies, covering about 5,000 people. One of the first duties the Council undertook was to seek the cooperation and support of the various Rhodesian branches of the British Medical Association. Negotiations resulted in the attainment of various benefits to our mutual advantage, including the important item of a specially reduced tariff of fees for all members and dependants of medical aid societies, in return for prompt payment of doctors' accounts. That the Representative Council has succeeded largely in the objects it set out to achieve is shown by the fact that the membership has grown to 22 societies, covering approximately 95,000 people.

The Group System, which is fast becoming a national feature of Rhodesia, and which is also intended to provide facilities for the formation of pension schemes, holiday associations, and other cooperative efforts, has been largely responsible for the rapid growth of medical aid insurance. It is estimated that, per head of population, there are more people enjoying health insurance benefits of this nature than anywhere else in the Commonwealth and, what is important, it is being achieved without State assistance or control.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following contributions to the Benevolent fund during October, November and December 1956, are gratefully acknowledged.

Votive Cards in memory of:

Dr. Arthur Stewart *by* Dr. R. A. Moore Dyke and Dr. F. L. A. Gace.
Mr. Dave Aberdien *by* Dr. Morris Cohen.
Mr. A. A. Miller *by* Dr. J. P. Immelman.
Mr. H. J. v. d. Heever *by* Dr. A. J. v. d. Spuy.
Mrs. McWhirter *by* Dr. and Mrs. S. H. Cohen.
Dr. R. C. J. Meyer *by* Transvaal Sub-Group Ophthalmological Society: Dr. A. J. Orenstein, Dr. A. W. Sichel, Dr. L. S. Robertson, Dr. E. Franks, Dr. H. Aneck-Hahn, Dr. M. Franks, Dr. L. Staz and Dr. R. Geerling.
Mr. Hugh Solomon *by* Dr. A. J. Orenstein and Dr. N. H. Kestell.
The Hon. Dr. W. E. Bok *by* Dr. R. Geerling.
Mrs. R. Rahder *by* Dr. and Mrs. R. Geerling.
Dr. Pringle *by* Border Branch.
Mrs. Burton *by* Border Branch.
Mrs. Burton *by* Drs. Cunard and Alexander.
Beryl le Page *by* Dr. and Mrs. R. Theron.
Dr. W. H. Herberg *by* Dr. and Mrs. R. Theron.
Hugh Warren Murdy *by* Dr. Morris J. Cohen.
Sir Lionel Whitby *by* Dr. A. J. Orenstein.
Mrs. H. R. Soloman *by* Dr. R. Geerling.
Mrs. Nell McWhirter *by* Natal Inland Branch.
Rev. Father Murphy *by* Dr. E. R. Hafner.
Dr. J. Jaffe *by* Dr. L. Ruskin.
Total amount received from Votive Cards £64 9s. 6d.

Services Rendered to:

V.C.M. *by* a Johannesburg Hospital Member.
Mrs. Dr. Harry Saacks and Son, Dennis *by* Drs. H. I. Maister, Russel, M. Neller and S. Abel.
Mrs. M. Snow and Joan Snow *by* Drs. N. Steere and F. Davidson.
Rev. R. F. G. Pearce *by* Dr. A. M. Whitaker.
Daughter of Dr. and Mrs. R. E. Stevenson *by* Dr. R. R. MacKenzie and Colleagues.
The Late Mrs. H. J. v. Rensburg, mother of Dr. B. J. v. Rensburg *by* Dr. D. J. de Villiers.
Rev. Johnson *by* Dr. H. Klevansky.
Mrs. Cilliers, wife of Dr. E. Cilliers *by* Dr. B. Knoblauch.
Dr. D. Vollet *by* Dr. J. A. Macfadyen.
Mr. and Mrs. F. H. Deppe *by* Dr. Buchan and Barlow.
A grateful patient *by* Mr. J. A. Curry.
Mrs. Mendel, wife of Dr. S. I. Mendel *by* Drs. S. Sacks and H. Grant-White.
Children of Dr. and Mrs. S. I. Mendel *by* Dr. F. Walt.
Dr. and Mrs. S. I. Mendel *by* Dr. M. R. Gitlin.
Miss N. S. Wilson *by* Drs. J. F. P. Mullins and Ware.
Dr. H. H. Golby and Family *by* Various Doctors.
Mrs. Leetion *by* Dr. McConnachi.
Mrs. D. S. Palmer *by* Dr. D. du Toit and Colleagues.
Mrs. P. G. Lange *by* Dr. G. S. Andrews.
Total amount received from Services Rendered £94 7s. 6d.

Donations:

	£	s.	d.
Cape Western Branch Collection Box ..	3	14	6
Dr. G. H. Caiger ..	5	0	0
Department of Anaesthesia of Groote Schuur	3	3	0
Cape Western Branch Collection Box ..	2	3	1
An Anonymous ..	9	9	0
Cape Western Branch Collection Box ..	4	0	7
Dr. D. McKenzie ..	3	0	0
Dr. G. F. C. Troskie	1	0	0
Proceeds of Annual Medical Ball 1956 S. Tvl. Branch ..	583	3	2
Proceeds from Jones Phillipson Cup Golf Competition ..	19	10	0
The following members of Natal Coastal Branch: Drs. E. W. S. Deale H. Grant-Whyte Alan B. Taylor Lance Knox Margaret Findlay A. C. Garnham F. Stern J. C. Baumann J. F. P. Mullins E. C. A. Fristedt	240	4	0
M. Byala L. V. Pearson R. Elsdon-Dew G. A. Drummond D. A. Edington J. D. F. Johnston R. C. J. Hill J. G. Bickerton J. J. Boulle B. Moshal B. C. Archer J. May F. R. Leonard F. Walt T. G. Armstrong R. H. Court Max Perlman			
Moira Soffé Max Gitlin H. L. Wallace M. J. Cohen J. M. C. Henderson J. D. M. McLaren E. B. Adams N. R. Butcher J. L. Treneman N. G. Steere T. Macleod K. W. Dyer H. R. Pooler Marjorie Bolton.			
Total £873 10s. 4d.			
Grand Total £1,032 7s. 4d.			

MINERS' CHEST CLINIC

Dr. A. J. Orenstein, M.D., F.R.C.P., Director, Pneumoconiosis Research Unit, has issued a notice on the following lines to benefit society doctors:

Under the auspices of the Pneumoconiosis Research Unit the 'Miners' Chest Clinic' will be opened in Johannesburg on Monday 16 September, in the grounds of the Johannesburg General Hospital, with an entrance direct from Hospital Street. The Clinic is intended to provide diagnostic and therapeutic services not commonly available to private practitioners, for cardio-respiratory diseases of European miners and ex-miners *only*. The services of the Clinic will be free of charge.

The physician in charge is Dr. G. K. Sluis-Cremer, who is not in private practice. The hours of consultation are:

Wednesdays, Thursdays and Saturdays ..	9 a.m. to 11 a.m.
Mondays and Fridays	3 p.m. to 7 p.m.
Tuesdays	3 p.m. to 5 p.m.
First Sunday only of each month	11 a.m. to 1 p.m.

Pulmonary disability may manifest itself before any radiological signs of silicosis can be observed. There is also sometimes no direct correlation between the degree of radiological abnormality and the degree of cardio-pulmonary disability. It is the particular purpose of this Clinic to provide diagnostic facilities for ascertaining as accurately as possible the cause and degree of the disability complained of, and to recommend treatment which might ameliorate, and even perhaps reverse or arrest, the morbid process.

The Pneumoconiosis Research Unit is seeking the cooperation of practitioners in this undertaking. When referring patients to the Clinic, doctors are asked to give each a note with any medical history in their possession. They will of course be fully informed of the findings and the proposed course of treatment, which will be carried out only with their concurrence.

NEW FOURTH SCHEDULE TO THE MEDICAL, DENTAL AND PHARMACY ACT (POISONS)

His Excellency the Officer Administering the Government has amended the Fourth Schedule to the Medical, Dental and Pharmacy Act by the substitution for the existing schedule of the following (Government Notice No. 216 of 26 July 1957):

FOURTH SCHEDULE : POISONS

DIVISION I

Alkaloids and Glucosides.—All poisonous alkaloids and glucosides, and their salts, not specifically named in this Schedule. Substances, preparations and admixtures containing in each single dose more than one-half of the maximum dose of the poison as shown in any of the recognized formularies declared under section 65 of the Medical, Dental and Pharmacy Act, 1928 (Act No. 13 of 1928).

Aconite alkaloids and substances, preparations or admixtures containing 1/50th% or more of alkaloids of aconite.

Apomorphine and substances, preparations or admixtures containing 1/5th% or more.

Atropine and substances, preparations or admixtures containing 1/10th% or more.

Belladonna alkaloids and substances, preparations or admixtures containing 1/10th% or more.

Brucine and substances, preparations or admixtures containing 1/5th% or more.

Calabar Bean alkaloids and their salts and substances, preparations or admixtures containing 1/5th% or more.

Coca alkaloids and substances, preparations or admixtures containing 1/10th% or more.

Cocaine and substances, preparations or admixtures containing 1/10th% or more.

Codeine and substances, preparations or admixtures containing 1/5th% or more.

Colchicine and substances, preparations or admixtures containing ½% or more.

Coniine and substances, preparations or admixtures containing 1/10th% or more.

Cotanine and substances, preparations or admixtures containing 1/5th% or more.

Digitalis, Digitalis glycosides and other active principles of digitalis unless diluted below one British Pharmacopoeia unit in each 2 grams.

Ecgonine and substances, preparations or admixtures containing 1/10th% or more.

Emetine and substances, preparations or admixtures containing 1% or more.

Ephedra alkaloids, salts of ephedra alkaloids; all synthetic substitutes for ephedrine, salts of all synthetic substitutes for ephedrine; substances, preparations or admixtures of the foregoing except preparations and admixtures containing not more than ½ grain per dose of ephedra alkaloids or synthetic substitutes for ephedrine in association with other medicinal substances.

Ergot alkaloids, salts of ergot alkaloids natural or synthetic, substances, preparations or admixtures containing the foregoing.

Gelsemium alkaloids and substances, preparations or admixtures containing 1/10th% or more.

Homatropine and substances, preparations or admixtures containing 1/10th% or more.

Hyoscine and substances, preparations or admixtures containing 1/10th% or more.

Hyoscyamine and substances, preparations or admixtures containing 1/10th% or more.

Jaborandi alkaloids and substances, preparations or admixtures containing ½% or more.

Lobelia alkaloids and substances, preparations or admixtures containing ½% or more.

Morphine and substances, preparations or admixtures containing 1/5th% or more.

Papaverine and substances, preparations or admixtures containing 1/5th% or more.

Pomegranate alkaloids and substances, preparations or admixtures containing ½% or more.

Sabadilla alkaloids and substances, preparations or admixtures containing 1% or more.

Solanaceous alkaloids not otherwise included in this division and substances, preparations or admixtures containing 1/10th% or more.

Stavesacre alkaloids and substances, preparations or admixtures containing 1/5th% or more.

Strophanthin and substances, preparations or admixtures containing ½% or more.

Strychnine and substances, preparations or admixtures containing 1/5th% or more.

Thebaine and substances, preparations or admixtures containing 1/5th% or more.

Veratrum alkaloids and substances, preparations or admixtures containing 1% or more.

Amidone (dl-2-dimethylamino-4: 4-diphenylheptan-5-one), its salts and any preparation, admixture, extract or other substance containing any proportion of Amidone.

Amidopyrine and its salts.

Antihistaminic substances, salts of antihistaminic substances, except when intended especially for the treatment of travel sickness.

Antimony potassium tartrate, antimony sodium tartrate, all substances, preparations and admixtures containing 1% or more.

Arsenic. Substances, preparations and admixtures containing 1/100th% or more of the equivalent of arsenic trioxide (As_2O_3).

Barbituric acid. Barbituric acid salts. Barbituric acid derivatives. Barbituric acid derivative salts. Substances, preparations and admixtures containing more than ¼% of any of the foregoing.

Beta-amino-propylbenzene. Alpha-methyl-penethylamine. Derivatives of both the foregoing having any group substituted by another radical. Preparations and admixtures of all of the foregoing, except when contained in appliances for inhalation in which the poison is absorbed in inert solid material.

Butyl chloral hydrate. Substances, preparations and admixtures containing 10% or more.

Cannabis indica and all solid preparations thereof except cannabis indica plasters.

Cantharidin. Substances, preparations and admixtures containing 1/100th% or more of the foregoing.

Chloral formamide.

Chloral hydrate. Substances, preparations and admixtures containing 10% or more.

Chloroform.

Cocaine substitutes, these being amino-alcohols esterified with benzoic acid, phenylacetic acid, phenylpropionic acid, cinnamic acid or the derivatives of these acids:

Orthocaine and its salts.

Oxycinchoninic acid. Derivatives of oxychinchoninic acid, and salts and esters of oxycinchoninic acid and its derivatives.

Para-aminobenzoic acid and its salts.

Para-aminobenzoic esters and their salts.

Phenetidylphenacetin.
Cocculus indicus (fish berries).
Cyanides of potassium and sodium. Hydrocyanic acid. Other poisonous cyanide substances, preparations and admixtures containing or yielding the equivalent of 1/10th% or more of hydrocyanic acid.
Diamorphine or diacetyl-morphine, also known as heroin, including all preparations, salts, admixtures or derivatives prepared therefrom or therewith and containing 1/10th% or more of diamorphine or of any derivative of diamorphine.
Di-isopropyl fluorophosphonate and substances, preparations and admixtures containing it.
Dinitrocresols, dinitrophenols, nitrophenols, dinitronaphthols, dinitrothymols, except substances not intended for treatment of human ailments.
Elaterin.
Fluoracetic acid. Fluoracetic acid salts. Substances, preparations and admixtures containing ½% or more of the foregoing.
Mercuric chloride (corrosive sublimate) and substances, preparations and admixtures containing 1% or more.
Nux vomica and all substances, preparations and admixtures containing 1/5th% or more of strychnine.
Opium and its alkaloids including morphine, and all salts and poisonous derivatives thereof and all substances, preparations or admixtures containing 1/5th% or more of anhydrous morphine or other alkaloid or poisonous derivatives of opium.
Para-amino-benzenesulphonamide. Salts of para-amino-benzenesulphonamide. Derivatives of para-amino-benzenesulphonamide having any of the hydrogen atoms of the para-amino group or of the sulphonamide group substituted by another radical, and their salts. Substances, preparations and admixtures containing the foregoing except those substances, preparations and admixtures thereof intended for external use.
Paraldehyde and preparations and admixtures thereof.
Pethidine, identified chemically as ethyl-1-methyl-4-phenyl-piperidine-4-carboxylate, its salts and derivatives and all preparations or admixtures containing 1/5th% or more of ethyl-1-methyl-4-phenyl-piperidine-4-carboxylate or its salts or derivatives.
Phenytoin (5 : 5-diphenyhydantoin), salts of phenytoin.
Phosphorous (yellow).
Phosphoros compound, the following:
Bis-dimethylaminophosphorous anhydride.
Bis-isopropylaminofluorophosphine oxide.
Diethylparanitrophenyl thiophosphate.
Diethylthiophosphate of ethyl-mercaptoethanol.
Dimethylaminofluorophosphine oxide.
Ethyl-paranitrophenyl-benzene thiophosphonate.
Hexaethyl tetraphosphate.
Hexaethyl thiophosphate.
4-Methyl-hydroxy-coumarin-diethyl thiophosphate.
Paranitrophenyl-diethyl phosphate.
Tetraethyl thiophosphate.
Triphosphoric pentadimethylamide.
Picrotoxin and all substances containing it.
Polymethylenebistrimethylammonium salts.
Radium.
Savin and its oil and substances, preparations and admixtures containing them.
Sulphonal and alkyl sulphonals, and preparations and admixtures containing them.
Thallium, its salts and acids.
Trichlorethylene.
Tridione (3 : 5 : 5-trimethyloxazolidine-2 : 4-dione).
Urethanes and ureides, all poisonous forms of.

DIVISION II

All substances, preparations or admixtures which are not included in Division I of this Schedule and contain a poison within the meaning of this Act, except substances, preparations or admixtures specifically excluded from Division II by the terms of this Schedule, and except substances to which section 82 of this Act applies.
All substances, preparations and admixtures containing more than 20% of chloroform.
Acetanilide and alkyl acetanilides.
Antihistamine substances intended specially for travel sickness.
Amyl nitrite.
Barium salts except barium sulphate.
Carbolic acid (phenol), cresylic acid (cresol), and all preparations containing 3% or more of any one, or of a mixture of these substances. (This includes 'lysol' and similar preparations under whatever name they may be described or sold).
Croton oil.
Essential oil of bitter almonds, unless free from hydrocyanic acid.
Lead acetate.
Lead plaster and its combinations (including machine-spread plasters), whether sold as diachylon or under any other name.
Methylacetanilide.
Mercuric iodide.
Mercuric thiocyanate (sulphocyanide).
Mercuric oxycyanide.
Mercuric oxides and substances, preparations and admixtures containing them, except if containing less than 3% of mercury.
Mercuric ammonium chloride (white precipitate).
Oxalic acid and its soluble salts.
Phenazone.
Rodenticides and similar preparations containing more than 1/20th% of [3-(-acetonylbenzyl)-4-hydroxycoumarin] under whatever name they may be described or sold.
Strophanthus.
Thyroid gland, dry thyroid, thyroid extract and active principles and derivations of thyroid gland.
Vermin killers. Substances, preparations and admixtures containing poisons prepared for the destruction of vermin if not included in Division I.

PASSING EVENTS : IN DIE VERBYGAAN

The Southern Transvaal Branch of the Medical Association have donated to the Benevolent Fund the proceeds of their annual medical ball, 1956, which amount to £583 3s. 2d.

* * *

The Natal Coastal Branch of the Medical Association have forwarded to the Benevolent Fund the sum of £240 4s. 0d. which was received for the Fund as the result of a collection amongst members of the Branch. The names of the subscribing members will be found on page 859 of this issue of the *Journal*.

* * *

Dr. L. J. A. Loewenthal, of Johannesburg, has returned from a visit to Europe during which he attended the International Congress of Dermatology in Stockholm.

* * *

Dr. Pierre F. M. du Toit, M.B., Ch.B. (Cape Town), M.R.C.O.G. (Lond.), M.O. & G. (Cape Town), has commenced practice as Obstetrician and Gynaecologist at National Mutual Buildings, Cape Town. Telephones: rooms 2-8414, residence 98-8691.

Dr. Pierre F. M. du Toit, M.B., Ch.B. (Kaapstad), M.R.C.O.G. (Lond.), M.O. & G. (Kaapstad) praktiseer tans as Verloskundige en Ginekoloog te National Mutual-gebou, Kaapstad. Telefone: kamers 2-8414, woning 98-8691.

* * *

Sir John Cockcroft has been appointed to preside over Unesco's Radio-Isotopes Conference in Paris, which is to be held on 9-20 September 1957. Sir John Cockcroft, who is a Nobel Prizewinner, is director of the Atomic Energy Research Establishment at Harwell, England.

* * *

Heese-Lawrence. The marriage of Dr. Margaret Lawrence, daughter of Brigadier and Mrs. C. Lawrence of Melbourne, Australia and Dr. Hans de Villiers Heese, son of Dr. and Mrs. J. de V. Heese of Stellenbosch, will take place on 31 August 1957 at the St. John's Presbyterian Church, London. Address, 76 Elgin Crescent, London, W. 11.

Dr. Ronald Singer, Senior Lecturer in Anatomy, University of Cape Town, has been awarded a grant by the Wenner-Gren Foundation for Anthropological Research, New York, to read two papers at the 5th International Congress of the International Association of Quaternary Research to be held in Spain in September 1957. He will also carry out research studies in the University of Barcelona.

* * *

The Workmen's Rehabilitation Centre, corner of Esselen and King George Streets, Hospital Hill, Johannesburg (telephone 44-9108), offers clerical services in connection with the completion of medical reports under the Workmen's Compensation Act. Medical practitioners may dictate the notes of their cases to a competent typist, or may use a tape recorder if necessary. The telephone services of the Centre may also be used to dictate notes on cases to typists at the Centre. The Centre will also undertake to forward completed medical reports if so desired. No charge is made for these services.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van ernstige epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk 2 Augustus—8 Augustus 1957.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	3	5	–	1	9
Kaapprovinsie ..	–	–	2	–	2
Oranje-Vrystaat ..	–	1	–	–	1
Natal	–	2	–	1	3
Totaal ..	3	8	2	2	15

Pes, Pokkies, Tifuskoors: Geen.

IN MEMORIAM

A. MARAIS MOLL, B.A., M.D., M.R.C.P.E.

Dr. A. Marais Moll who died on 16 July 1957 at Mouille Point, Cape Town, was born at Paarl, Cape Province, on 31 July 1893. He attended the Boys' High School, Paarl, and graduated B.A. at Cape Town University, where he also took the earlier part of his medical course. He then proceeded to Edinburgh University, where he qualified M.B., Ch.B., in 1919. Before leaving South Africa he had served in the South African forces in World War I, and after qualifying he held a commission in the R.A.M.C., serving in Palestine.

A. Marais Moll

After the war Dr. Moll was engaged in general practice at Paarl for 9 years, and then returned to Edinburgh, where he took his doctorate with honours in 1932, in which year he was also admitted M.R.C.P. (Edin). Returning to South Africa as a consultant he was appointed to the honorary medical staff of the Somerset Hospital, Cape Town, and moved to Groote Schuur Hospital when the transfer took place in 1938, and later became honorary Physician to the hospital. For some years Dr. Moll was chairman of the Medical Advisory Subcommittee of Groote Schuur Hospital, which position he held during the change-over from the honorary system. He was a member of the University Council and Senate. On his retirement from the hospital he served for 7 months as Acting Dean of the Faculty of Medicine of the Cape Town University, in which capacity he served on the Central Hospitals Committee of the Cape Province.

Dr. Moll had wide interests outside the medical world. He was M.P. for Rondebosch from 1938 to 1948, and served on the National Health Commission under the chairmanship of Dr. Henry Gluckman, M.P. One of his great interests was the Rustenburg High School for Girls, where his daughters attended. He was a member of the School Committee of the Rustenburg Junior and High Schools from 1939 (with a break in 1942-43) and chairman from 1945 to 1957. Miss Thomson, the Principal of the school, speaks in the warmest manner of the great services he gave to the school. He was extremely interested in this work, and his departure was regarded as 'an irreparable loss to the school'. He was 'a magnificant chairman', able in guidance, strong in support, and with a faculty of disagreeing without arousing rancour. The cordial relations between the school and the school committee were largely due to his personality.

Prof. F. Forman, of Cape Town, writes: With the death of Dr. A. Marais Moll there passed away one of the old-type physicians whose loss will considerably impoverish our lives. During his years of general practice in Paarl he was the 'beloved physician' and the 'compleat' doctor because of his understanding of people and human affairs. When, ripe with experience, he became a specialist physician he joined the staff of the teaching hospital of the Medical School of the Cape Town University, and was soon head of one of the medical units. On reaching the age-limit, he retired from this post as well as from medical practice, but continued to serve his old alma mater on both the University Council and Senate. When he was chairman of the Medical Advisory Committee of the Groote Schuur Hospital the meetings were conducted superbly. At all times Dr. Moll could be depended upon to uphold the cause of justice, fearlessly and courageously. He was a fighter—but always without malice or personal animosity.

Dr. Moll was a many-sided personality and was widely knowledgeable. He had a remarkable capacity for friendship.

Dr. P. Leftwich, of Cape Town, writes: To all who were associated with him, colleagues, students and patients, Dr. Marais Moll has left behind a memory of a vital, jolly and happy man. He was a 'character' and his most outstanding quality was the possession in exceptional degree of the human touch in his social and professional contacts. A true *bon vivant*, he loved company, and his sense of fun and his intense sociability made him the popular centre of any group in which he found himself; there was never a dull moment when he was near. He loved life and could never resist a party, and I look back with pleasure at the generous hospitality to be found in his home on many occasions. Although in poor health for many years, this did not damp his spirits or his sense of fun.

It was as head of a medical firm at Groote Schuur Hospital, Cape Town, that I came to know him best and to learn to admire his unusual approach to the routine of work in the wards. A sound clinician of the old school, he brought more than mere technical ability to his handling of patients. Medicine was an art for him, and because of this and his warm-hearted outlook he practised it by applying a highly personal approach to the patient's problems. He never lost the attitude of mind of the family doctor and he sensed only too well the secret fears and anxieties that so often consumed the patient, separated from home and family. He made a point of seeing patients daily, and always had a genial greeting or a teasing remark for each of them. Every patient felt that he was being singled out for special care and attention, and his sunny disposition was infectious and seemed to bring a

holiday spirit into every ward. Members of his firm will remember his taking an elderly African patient on his first visit to the sea, and the simple-minded countryman from the isolated 'die Hel', whom he escorted to the shopping centre of the city. It was not surprising that he earned the enduring affection of patients of all races for years after their discharge from hospital.

He was well known as a first-class after-dinner speaker, humourous and witty, and an asset at dinners and banquets, and his long Parliamentary experience made him a valuable chairman and member of hospital and University committees over many years.

It was a privilege to have known this human and lovable man.

Dr. the Hon. Henry Gluckman, M.P., formerly Minister of Health, writes: Dr. Moll will be remembered for the great contributions which he has made, not only in connection with the advancement of medicine in South Africa, but also for his fine record as a Member of Parliament. Others are more fitted to pay adequate tribute to his work as a leading physician. It is my privilege to place on record the place which he carved out for himself as a Member of Parliament.

His profound and extensive medical knowledge enabled him to make authoritative statements on medical and social problems affecting all sections of the people of the Union of South Africa. He endeared himself to his colleagues because of his charm and friendliness. He was sadly missed when he was not returned to Parliament and his parliamentary colleagues will now mourn his death.

I, personally, was deeply beholden to him for his ever-ready assistance and guidance as a member of the National Health Services Commission, of which I had the honour to be Chairman. Throughout a period of 2 years, during which we travelled the length and breadth of this country, and took evidence from hundreds of individuals and organizations, my friend the late Dr. Moll was there to sort evidence, to elicit information, to collect, to collate and finally to present that which was relevant for inclusion in our recommendations.

All those who have known him in his various spheres of activity will, I know, join me in extending to his wife and family, our deepfelt sympathy in their grievous bereavement.

* * *

DR. W. E. MCLELLAND

J. Carlyle-Mitchell, of Durban, writes: William Edward McLelland was born at Alexandria, Dumbarton, Scotland, on 1 June 1884, the son of Dr. Alexander McLelland of that town. Educated at Blair Lodge School, Perthshire, he commenced studying at Glasgow University, but on account of ill-health he was compelled to abandon his studies and came to South Africa, where for a time he took up farming. Later, and in much better health, he went to Australia, where he decided to resume his life's work, and he finally qualified as M.B., B.S. at Melbourne University.

Dr. W. E. McLelland

Returning to South Africa, he first practised at Witbank in the Transvaal, and later settled at Nottingham Road, Natal, where he was the medical officer for many years at Michaelhouse, Balgowan, and King's Preparatory School, Nottingham Road, and he is affectionately remembered by many boys of those schools.

A man of wide interests, especially in art and literature, he will also be remembered as the breeder of the well-known Ardenlee strain of Rhode Island Red poultry. He won many high awards with his birds in the show ring.

His death occurred suddenly on 17 April 1957 and he was interred at Mountain Rise, Pietermaritzburg. He is survived by his wife, Mrs. Olive McLelland.

REVIEWS OF BOOKS : BOEKRESENSIES

THE DOCTOR AS A WITNESS

The Doctor as a Witness. By John Evarts Tracy. Pp. xiv + 221. $4.25. Philadelphia and London: W. B. Saunders Company. 1957.

Contents: I. The Doctor is Called as a Witness in a Legal Proceeding. II. The Privileges and Obligations of the Doctor-Witness. III. Opinion Evidence. Expert Testimony. IV. Direct Examination of the Doctor-Witness. V. Cross-Examination. VI. Testimony on the Issue of Insanity. VII. Testimony in Workmen's Compensation Proceeding. VIII. Testimony in Malpractice Cases. IX. Preparation for Trial or Hearing. X. What Makes a Good Medical Witness. XI. The Compensation of the Doctor-Witness. XII. Proposed Improvements in the Law as to the Use of Expert Medical Testimony. Index.

This is an ideal book for any doctor whose professional duties bring him to the courts as a witness. It is simply and clearly written in an easy style, and is easy for the layman in law to follow. There are no voluminous references to case law, which only disturbs the lay reader and makes him more afraid of the law than ever.

The book does not by any means limit itself to the law of evidence. It deals with all aspects of a trial where the doctor is the most important witness. As such, it covers court procedure and also mentions the issues which became paramount in medico-legal work.

Being written for the American public, there is constant reference to the jury. South African practice does not know juries for civil trials, so that the South African reader must mentally substitute 'judge' for 'jury'.

In a simple manner the author deals with commonplace legal situations which the doctor may encounter. He takes the doctor through all the phases of his examination in the witness box, giving sound practical advice. Of all the advice perhaps the most important is that a doctor should never identify himself with one party or the other. A medical witness must present his facts in a scientific objective manner. He is in court as a trained observer. If he sticks to his medical observations his cross-examination will be reduced to a minimum. The doctor who feels he must help the prosecution to secure a conviction is allowing zeal to overcome objectivity, and will only expose himself to heavy cross-examination. Particularly in criminal cases must the doctor remember that on the medical testimony a man may lose his liberty. One can never be too careful in aiming at the highest degree of accuracy in giving medical evidence.

Professor Tracy indicates that he has derived much pleasure from writing this book. The reader will likewise find it a pleasure, and in addition will derive much useful information from it.

A.P.

CORRESPONDENCE : BRIEWERUBRIEK

THE PAROTID SALIVARY GLAND

To the Editor: I was interested in the excellent article on the parotid gland by Mr. D. J. du Plessis[1] in the *Journal* of 3 August 1957.

In the section on congenital sialectasis he says this may be due to an underlying congenital defect as yet undetermined. He shows a photograph of such a patient in which it is obvious to me that the congenital defect is due to pressure of the bony pelvis (probably the sacral promontory) on the unborn foetus. This is

brought about by unusually early descent of the foetal head several weeks before term. It commonly causes some flattening and thinning of the posterior ear. Less commonly it causes congenital torticollis or facial defects such as is shown in a photograph which was reproduced in an article on congenital torticollis.[2]

In cases where the head has engaged unusually early and deeply, I always warn the expectant mother that the posterior ear will be thin and flattened, and I look for evidence of congenital torticollis as soon as the baby is born.

This rather obvious aetiology of congenital torticollis and associated conditions has not yet come to be generally known by surgeons.

G. P. Charlewood

1104/5 Medical Centre
Jeppe Street
Johannesburg
8 August 1957

1. Du Plessis, D. J. (1957): S. Afr. Med. J., **31**, 773.
2. Charlewood, G. P. (1947): J. Obstet. Gynec., **55**, 500.

ALKAPTONURIA

To the Editor: I read the article on two cases of Alkaptonuria by Politzer and Reitz[1] appearing in the *Journal* of 3 August 1957, with great personal interest for I have been searching for such cases ever since I first discovered a patient with alkaptonuric arthritis in Johannesburg, which I reported in full in the *Journal of Bone and Joint Surgery*, Vol. 33B. No. 3 in August 1951.

As far as I know, these are now the only three cases to have been reported from South Africa, but I feel sure that there must still be some patients with alkaptonuria in this country in whom the diagnosis has been missed, even though the incidence of the condition is supposed to be as low as one in ten million.

I would suggest that in every case of chronic arthritis the urine be examined and a search made for ochronosis in the sclera and the ears, and I would appreciate a report from any practitioner who has knowledge of any other cases of alkaptonuria in South Africa.

Sidney Sacks

1010 Medical Centre
Jeppe Street
Johannesburg
8 August 1957

1. Politzer, W. M. and Reitz, C. J. (1957): S. Afr. Med. J., **31**, 782.

NUFFIELD BIRTHDAY FUND

To the Editor: On 10 October next, Lord Nuffield celebrates his 80th birthday. As is well known, Lord Nuffield is the greatest benefactor to medicine the Commonwealth has ever known, and there can be few medical men and women who have not benefited, directly or indirectly, from his gifts.

It seems fitting to us that, on the occasion of his 80th birthday, members of the medical profession should have an opportunity of showing the regard which they have for Lord Nuffield, and their gratitude for his farsighted generosity.

With this in mind, we write to ask members of the profession to contribute, however modestly, towards a birthday present which we propose to give Lord Nuffield as a tribute from the profession, both in the United Kingdom and the Commonwealth, at some celebration to be held in London or in Oxford near the date of his birthday.

It is our intention to buy a present for Lord Nuffield from the money subscribed, and to give him the balance to use as he wishes.

A maximum donation of about two guineas is suggested, but any amounts, either larger or smaller, will be very acceptable. Donations of even a few shillings would be welcome, for the important thing is that Lord Nuffield should know that the present comes from the profession as a whole.

We earnestly ask you to send a contribution.

Cheques and postal orders should be made payable to the Nuffield Birthday Fund, and sent to 1 Wimpole Street, London, W. 1, England, as early as possible.

1 Wimpole Street
London, W. 1
England
August 1957

W. Russell Brain
Henry Dale (*Trustee*)
Evans
R. R. MacIntosh
W. N. Pickles
Harry Platt
Arthur Porritt
Charles D. Read
Clement Price Thomas (*Trustee*)

'N NUWE BENADERING VAN DIE VRAAGSTUK VAN PROSTITUSIE

Aan die Redakteur: Daar is seker geen ander openbaring van ons maatskaplike patologie wat die sosiale gewete meer gaande gemaak het as prostitusie nie, en ondanks die ywer waarmee beheermaatreëls toegepas word, is hierdie vraagstuk nog net so omvangryk soos voorheen. Dit is egter nie moeilik om die rede hiervoor te vind nie. Die feit dat ons nog nie daarin geslaag het om hierdie vraagstuk in die samelewing op te los nie, is grootliks te wyte aan ons verkeerde opvatting van die menslike natuur. Daar kan dus aangeneem word dat, alvorens ons die mens as 'n psigo-somatiese wese beskou wat onafskeidelik aan sy veelvormige omgewing verbonde is, ons nie in staat sal wees om die uiteenlopende faktore wat meewerk om sy persoonlikheid te ontwrig, te begryp nie. Hierdie ontwrigting van die individu se persoonlikheid openbaar gewoonlik sig in die een of ander afwykende vorm van gedrag, soos bv. prostitusie, dranksug, misdadigheid, verslaafdheid aan verdowingsmiddels, en psigo-neurotisisme. Hierdie afwykende gedragsvorms staan egter nie los nie, en kan dus nie afsonderlik behandel word nie; hulle is eerder onderling afhanklike openbarings van die faktore en kragte wat die ontwrigting van die mens se persoonlikheid bewerkstellig. Hierdie ontwrigtende faktore kan in die fisiese òf in die psigiese gedeelte van die mens se persoonlikheid geleë wees, maar is egter merendeels milieu-faktore wat op die individue waaruit die sosiale organisme saamgestel is, inwerk. 'n Ontwrigtende faktor kan omskryf word as 'n faktor wat die mens sy veiligheid op sielkundige, geestelike of ekonomiese gebied ontneem.

Hierdie faktore kan te enige tyd in die gemeenskap werksaam wees, en staan buite die beheer van die individu. Grootskeepse werkloosheid wat tydens ekonomiese depressie voorkom, lei by voorbeeld tot ekonomiese ontbering wat op sy beurt weer aanleiding gee tot die sielkundige verskynsel van bekommernis wat by die individu die oorweldigende drang opwek om aan die ondraaglike sosiale omstandighede te ontvlug.

Die jong meisie wat dus bv. ekonomies en geestelik ontroof is deur die dood van een of albei ouers; deur die egskeiding van haar ouers; deur verlating deur een van die ouers, of omdat sy ver van haar ouers af is, en geestelike aanraking met hulle moet ontbeer—so 'n meisie sal vroeër of later, ten einde siel en liggaam aanmekaar te hou, swig voor versoekings wat haar uiteindelik sal meesleur op die weg na prostitusie, drank, en misdaad.

Dit is duidelik dat die oplossing van die probleem van prostitusie in ons gemeenskap, afhang van ons bereidwilligheid om in die eerste instansie daardie faktore wat in die maatskaplike omgewing aanwesig is, en wat bydra tot die ontwrigting van die gemeenskap aan die een kant, en die ontwrigting van die gesin aan die ander kant, uit te skakel.

Ten slotte wil ek egter sê dat daar behoorlike erkenning verleen moet word aan die mag van die godsdienstige drang in die individu.

Ek het hierdie drang omskrywe as die integrerende drang wat God in die siel van elke wese geplaas het, en ek wil beweer dat, indien hierdie drang deur haat of vrees verdring is, dit weer deur liefde en begrip opgewek kan word. Die meeste mense moet egter liefde van buite af kry. Die individu moet liefde van sy gesin ontvang, en die gesin weer van die gemeenskap, en die maatskaplike aarde moet vrugbaar gemaak word sodat die liefde wat die individu en sy gesin, asook die gesin en die gemeenskap saambind, daar kan groei.

Louis F. Freed

Barbican-gebou 2
Presidentstraat
Johannesburg
14 Augustus 1957